# GOUT,

## AND ITS RELATIONS TO DISEASES OF THE LIVER AND KIDNEYS

BY

ROBSON ROOSE, M.D.

*SEVENTH EDITION*

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BY

ROBSON ROOSE, M.D., LL.D., F.C.S.

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TO

THE HONOURED MEMORY

OF

**SIR WILLIAM W. GULL, BART., M.D. LOND., F.R.S.,**

D.C.L. Oxon., LL.D. Cantab. and Edin.,

TO WHOM

THE PREVIOUS EDITIONS OF THIS WORK

WERE BY PERMISSION

Dedicated,

AS A TOKEN OF GRATITUDE, ESTEEM, AND ADMIRATION.

## PREFACE TO THE SEVENTH EDITION.

SEVERAL years having elapsed since the publication of the last edition of this work, which has been for some time out of print, I have subjected its pages to a thorough revision. I have also thought it right to discuss in fuller detail various manifestations of gout which had been previously dealt with in a few sentences, inasmuch as they were not included within the limits defined by the title of the work. The expansion is, I trust, excusable as tending to develop and reinforce the theories which I have striven to establish. Other additions embody the results of my recent experience with regard to the treatment of gout and its multiform complications. I have also consulted the recent works of other authors, and have alluded to such facts and views as I considered important. I trust that nothing has been omitted that deserved mention; and I can only add that if my work is not better calculated than at any former time to fulfil the purposes for which it was written, the fault must not be ascribed to want of endeavour.

45, HILL STREET,
BERKELEY SQUARE, W.
*June*, 1894.

# PREFACE TO THE FIRST EDITION.

THE following pages contain the results at which I have arrived after a somewhat extensive clinical experience of the gouty dyscrasia in its various forms. While far from denying the influence of heredity and other causes, I am fully convinced that functional disorder of the liver underlies the majority of gouty manifestations, and that the kidneys are only secondarily implicated. If this view be correct, it would seem to follow that the object of treatment should be not merely the neutralisation of the *materies morbi* by means of alkalies, but, in an especial manner, the restoration of the hepatic functions. How this is to be effected, I have described at some length in the chapter on treatment. All statements quoted from other writers have been carefully criticised, and I have endeavoured to make my book in every way a record of my own observation and experience.

45, HILL STREET,
BERKELEY SQUARE, W.
*May*, 1885.

# CONTENTS.

## CHAPTER III.

### Theories as to the Nature of Gout.



## CHAPTER IV.

### Causes of Gout.



## CHAPTER V.

### Irregular Manifestations of Gout—Visceral and Cutaneous Affections.

## CHAPTER VI.

### Hepatic and Renal Disorders Connected with Gout.

## CHAPTER VII.

THE TREATMENT OF GOUT AND OF VARIOUS DISORDERS CONNECTED WITH IT.

# GOUT,

## *AND ITS RELATIONS TO DISEASES OF THE LIVER AND KIDNEYS.*

## CHAPTER I.

### GENERAL CONSIDERATIONS WITH REGARD TO GOUT; THEORIES AS TO NATURE, AND MORBID APPEARANCES.

PECULIARITIES OF THE DISEASE—VIEWS AS TO NATURE AND RELATIONS TO DISEASES OF LIVER AND KIDNEYS—DIATHESIS, DEFINITION OF—MEANING OF THE TERM GOUT—CULLEN'S DEFINITIONS AND CLASSIFICATION—VISCERAL AND ESPECIALLY RENAL SYMPTOMS—THEORIES AND FACTS TO EXPLAIN NATURE OF GOUT—SODIUM URATE IN THE BLOOD—SIR W. ROBERTS' VIEWS—OTHER CHANGES IN THE BLOOD—CONDITION OF THE URINE—MORBID APPEARANCES IN THE JOINTS, AORTA, HEART, LUNGS, AND KIDNEYS.

GOUT is undoubtedly one of the most remarkable and perplexing disorders with which the physician has to deal. Wayward and capricious in its onset and course, and astonishingly varied in the forms which it assumes, its manifestations may at first sight appear to be subject to no intelligible law. An acute attack, indeed, is marked by a perfectly definite series of phenomena, as regular in their development as

those of uncomplicated scarlatina. On the other hand, the disease may be almost entirely latent, its presence being revealed by obscure symptoms, slight in degree, and evanescent in character, and often referable to their true origin only when a decided family proclivity, or the subsequent progress of the case serves to guide the diagnosis aright. Midway between these forms are others in which the malady makes itself felt either in the internal organs alone, or in these alternately with the superficial parts. Thus, while in certain instances the symptoms may be indicative solely of functional disturbance or of structural mischief in the kidney, liver, or stomach, in others they alternate with external phenomena, such as pain and swelling in one or more of the joints. The study of an affection so complex, and, if I may be allowed the expression, kaleidoscopic in its character, is full of interest, but it obviously presents considerable difficulty.

It is not my intention to discuss in detail the ordinary symptoms of gout, for they are too well known to need description. I shall refer only to such of its features as are more immediately connected with that theory of its pathology which I am anxious to establish. My object in this little work is to place

before the profession my views as to the nature of gout, and more especially as to its relations to those disorders of the liver and kidneys with which, according to my experience, it is very often connected. I shall also add a few words on those affections of the skin which I believe to be due to the presence of the gouty diathesis. I use the word "diathesis" to express "a general tendency, in virtue of which an individual becomes the subject of several local affections, similar in their nature." I believe that gout is a disease of a *specific* character, but capable of assuming widely different forms, and of causing a vast number of symptoms, disturbances and complications, many of which often remain inexplicable until an acute attack furnishes the key to the diagnosis.

There can be no objection to the use of the word "gout" to designate the disease. Its meaning is well understood, and it is much to be preferred to "podagra," "chiragra," etc., which merely indicate some of the local manifestations. The term "gout," on the other hand, owed its origin to the "humoral" view of the pathology of the disease, the idea being that some morbid humour existed in the blood, and was thrown out or distilled into the joints

"drop by drop." We shall presently see that this idea is the basis of that theory of the origin of the disease which is most in accordance with well-ascertained chemical and pathological facts.

If we wish to define the word "gout," and to classify the various forms of the affection, we can scarcely do better than adopt Cullen's language and arrangement. Cullen tells us that gout is hereditary, coming on without apparent external causes, but generally preceded by an affection of the stomach; that it is characterised by pyrexia, by pain at some one of the joints, generally at that of the great toe; certainly attacking by preference the articulations of the feet and hands; returning at intervals, and often alternating with affections of the stomach or other internal parts. *Regular* gout, Cullen defines as characterised by considerable and violent inflammation of the joints, continuing for several days, and receding gradually with swelling, itching, and desquamation of the affected part. His second variety is *atonic* gout, in which there is an affection of the stomach or of some other internal part, either without the usual inflammation of the joints, or with slight and transient pain in them, often suddenly alternating with dyspepsia or other

symptoms. In the third form, *retrocedent* gout, the inflammation of the joints suddenly disappears, but is succeeded by disorder of the stomach, or of some other internal part. In *misplaced* gout, "the gouty diathesis, instead of producing the inflammatory affection of the joints, produces an inflammatory affection of some internal part." Cullen adds to these descriptions the significant remark that gout is sometimes accompanied by other diseases. This old nomenclature is really based on clinical observation, and does not involve any theory as to the nature of the disease. Cullen's opinion* on this point, however, was that "gout is manifestly an affection of the nervous system."

Whatever form the attacks of gout may assume, other symptoms are pretty certain to become developed during the course of the disorder, and especially in the intervals between the attacks; of these, hepatic derangements, dyspepsia, and gravel are by far the most common. If, moreover, careful inquiry be made into the history of a case of acute gout, we almost always find that the attack has been preceded by more

* "Cullen's Works," edited by John Thomson, M.D., 1827, Vol. ii., p. 122.

or less distinct symptoms of disorder of the liver or stomach, or of both of these organs; and this fact is of importance as regards the etiology of the disease. There is another well-marked characteristic of gout, viz., that in most cases the attacks become more frequent year by year, without diminishing in length; the disease in many cases being really continuous, but presenting irregular exacerbations and remissions. This chronic stage generally owes its origin to serious implication of the kidneys.

Three or four principal theories are still in vogue with regard to the nature and origin of gout. According to the first of these, gout is due to digestive anomalies, causing the blood to become loaded with certain morbid elements produced mainly in the stomach and duodenum, and uniting in the blood with some element of the bile which has been suffered to accumulate through defective secretory action of the liver. This view was supported by the late Dr. Todd,* who admitted, however, that the condition is usually associated with the lithic acid diathesis. The second theory likewise assumes an impure state of

* 'Practical remarks on Gout, Rheumatic Fever, etc.," p. 74.

blood, but refers this condition principally to disturbance of the renal function. Sir A. Garrod is the chief supporter of this theory. The third view, that of Cullen, already referred to, has lately been resuscitated. It assumes that the nervous system is primarily at fault in cases of gout. Another theory has been recently advanced by Dr. W. F. Wade,* of Birmingham, who, while admitting the existence of a blood-dyscrasia, thinks that "in gouty arthritis, the joint-affection is secondary to and caused by a preceding (though possibly only by a very short time) affection of the peripheral nerves." He also contends that a *local nerve-theory* of gout explains the immense preponderance of attacks in the tarso-metatarsal joint of the great toe.

Putting theories on one side for the present, let us see what *facts* we possess which may serve to explain the nature of gout. The most important *fact* in connection with the pathogeny of gout is that uric acid is found in the products of gouty inflammation, and that the blood contains an increased amount of this substance, which in both cases is combined with soda. In

* "On Gout as a Peripheral Neurosis," 1893.

health, only the most minute traces of uric acid can be detected in the blood; in gout, more than one-sixth of a grain has been obtained from 1000 grains of serum. The process for its detection, as devised by Sir A. Garrod, is simple enough. One or two fluid drachms of serum are put into a flattened glass-dish or capsule, three inches in diameter, and about one-third of an inch in depth; six minims of strong acetic acid are added to each drachm of serum. The fluids are well mixed, and a few fibres of linen are introduced by means of a glass rod. The glass is then set aside in a cool place until the serum is nearly dry, and if uric acid be present it will crystallise in a rhombic form on the threads, and will be easily recognised under the microscope. The serum must be fresh, for otherwise the uric acid will undergo decomposition and become converted into oxalic acid and urea. When blood cannot be obtained, the experiment may be performed with the serum collected from a blister, provided that this latter has not been applied to a point attacked by a gouty inflammation, inasmuch as inflammatory action causes the uric acid to disappear from the affected part and its immediate neighbourhood. In cases of chronic gout, uric acid can always be detected in the

serum; in acute gout it may be absent between the attacks, but can always be discovered shortly before these take place. It must be borne in mind, however, that a similar excess of uric acid in the blood is often found in cases of chronic lead-poisoning and of certain diseases of the kidney. Besides being found in the blood, uric acid often exists in various secretions and fluids in gouty cases. Thus it has been found in the cerebro-spinal fluid; in the effusion of pleurisy and pericarditis; in intestinal secretions; in the discharges from cutaneous eruptions, and in the form of dust (sodium urate) on the skin.

According to Sir W. Roberts,* who has very carefully investigated the chemistry of uric acid and gout, in the latter disease "uric acid is thrown down in a state of combination as sodium bi-urate in the interior tissues of the body." The precipitated substance acts as a foreign body, and tends to set up inflammatory processes in the implicated parts, and the manifestations of gout are thus proximately due to mechanical injury. Sir W. Roberts has proposed the term "uratosis" as signifying the deposition of urates in

* "Croonian Lectures, 1892, on the Chemistry and Therapeutics of Uric Acid, Gravel, and Gout."

the tissues. Under normal circumstances uric acid exists in the urine, and probably also in the blood, as a quadri-urate of potassium, sodium, and ammonium. The sodium-bi-urate, which constitutes the deposit in gout, is a pathological combination, and is far less soluble than the quadri-urate. In a state of health, the elimination of the latter "proceeds with sufficient speed and completeness to prevent any undue detention or any accumulation of it in the blood. But in the gouty state this tranquil process is interrupted, either from defective action of the kidneys, or from excessive introduction of urates into the circulation, and the quadri-urate lingers unduly in the blood and accumulates therein." It subsequently becomes transformed into the bi-urate, at first in the hydrated or gelatinous form, but afterwards in the almost insoluble anhydrous or crystalline condition.

The blood presents other, though far less easily discoverable differences in its chemical composition. These are summed up by Charcot* as follows:—

1. In acute gout the proportion of the corpuscles is not diminished (in this the disease differs

* "Lectures on Senile Diseases," New Syd. Soc. Transl., p. 55.

remarkably from rheumatism); in chronic gout there is considerable diminution and more or less anæmia.

2. In acute gout the fibrin is increased; the clots are buffy.
3. In chronic gout the albumen of the blood is diminished, if there be any disease of the kidneys, and in such case the blood contains an increased amount of urea.
4. The alkalinity of the blood is diminished, a condition which seems to favour the production of chalky deposits.
5. The blood sometimes contains traces of oxalic acid.

It is evident from the foregoing statements, that while excess of uric acid is the most prominent and characteristic change in the blood in gout, other alterations, more or less important, are frequently present.

The condition of the urine in gouty subjects exhibits several points of interest, which may be thus briefly enumerated. During acute attacks the secretion is scanty and high-coloured, but it contains less than the normal quantity of uric acid; perhaps only two grains, instead of eight or nine, are excreted in the

twenty-four hours. The blood-serum can easily be shown to contain excess of uric acid. In the intervals the condition of the urine varies, but there are often deposits of urates and of crystalline uric acid. In chronic gout the excretion of uric acid is for the most part diminished, but from time to time there are crystalline and amorphous deposits containing this substance. Oxalate of lime is often found, tube-casts and albumen are by no means rare, and sugar is occasionally present.

Having thus briefly referred to the state of the blood and urine, the morbid changes in gouty subjects constitute the next point of importance. They are well marked in severe and chronic cases, and are for the most part the direct consequences of the excess of uric acid in the blood. Certain tissues and organs are the seat of deposits consisting of sodium urate, and a careful study of these changes throws much light upon the nature of the disease. The existence of the so-called "chalk-stones" has been recognised from a very early period, but they were regarded as associated only with very severe cases of gout. A closer investigation has shown that even a very slight attack leaves behind it marked changes in the

structure of the joints, and that these changes generally remain, even in favourable cases, throughout life. The cartilages of the joints are the parts primarily involved; deposits of sodium urate form in them, and, as pointed out by Charcot, occupy the superficial part, and are situated either between the cells, or actually within them. The first changes generally take place as far as possible from the insertion of the synovial membrane—a fact which Charcot explains by assuming that the so-called non-vascular tissues are especially liable to be the seat of these deposits. It may be that urates are less soluble in lymph than in blood. On the other hand, the articular surfaces of the bones and the synovial membrane, being highly vascular, are less liable to be thus invaded.

At a later period of the disease, a similar deposit is found in the synovial membrane, and here the same rule is observed, inasmuch as the less vascular portions at the circumference are the first to be affected. As the disease progresses, the ligaments, the tendons, and the connective tissue surrounding the joint are successively attacked. Sir W. Roberts alludes to the close and special connection between the synovial fluid and gouty deposits. The former "has itself been

repeatedly found heavily laden with crystals of sodium bi-urate." The synovial fluid is rich in sodium salts, the presence of which appears to favour the deposit of urates. It very rarely happens that deposits of this kind are found in the substance of the bone itself, owing, it may be presumed, to the marked vascularity of the osseous tissue. Even where the surfaces of the cartilage are completely encrusted, the neighbouring portion of the bone is usually free from any trace of uric acid. The bones of gouty persons, however, frequently contain an increased amount of fat and a diminished quantity of earthy matter. The most important point in connection with these deposits is, that they appear to avoid the neighbourhood of the blood-vessels. The cartilage of the ear is often thus affected, and in a few rare cases which have been reported the nodules were visible in this part alone, while on post-mortem examination the ligaments and cartilages of many articulations were found to be more or less thickly covered. In still rarer cases no peculiar changes in the joints are discoverable after death ; but it is reasonable to assume that in these instances the uratic deposits had been dissolved. It is a well-known fact that tophi on the pinna occasionally disappear.

In acute attacks of gout the metatarso-phalangeal joint of the great toe is the one most commonly affected. This joint was affected in 373 out of 512 first attacks recorded by Sir C. Scudamore. Next to it in order of frequency come the fingers, and then the knees and elbows; other joints are liable to suffer, but the morbid changes are very seldom so severe as in those just enumerated. It not unfrequently happens that the great toe is the only joint attacked. Several reasons have been adduced to account for this preference; it has been supposed that this joint, bearing, as it does, much of the weight of the body, and often cramped by ill-fitting boots, is peculiarly liable to injury, and is therefore a *locus minoris resistentiæ*.* This theory is supported by those instances in which injuries to joints in gouty subjects have been followed by the characteristic deposits. A case has been reported to me by a friend, of an extremely gouty subject, who took very little exercise save on horseback. In his case, during a long series of years, the knees alone were affected. Similar results of injuries to

* The influence of cold is probably instanced in the case of the external ear. The joints appear liable to gout very much in direct proportion to the severity of the mechanical wear and the changes of temperature they are subjected to. Wilks and Moxon, "Pathological Anatomy," page 80.

bones and joints often occur in the course of syphilis. Dr. Wade supports his local nerve-theory (see page 7) of gout by various anatomical considerations. He states that "the nerves around the great toe-joint are not deeply seated in the soft parts; they are subject to pressure on the sole aspect by the weight of the body, on the inner and upper aspects by the upper leather of the shoe. The nerves of the other toes which are rarely attacked are protected by the soft parts and by the cushion of the sole, and are in most boots much less pressed upon than the great toe by the upper leather."

This deposit of sodium urate is peculiar to gout, and does not occur in any form of rheumatism. It is, as Sir A. Garrod points out, the *cause* and not the *effect* of the inflammatory symptoms, for when these are severe the urate undergoes alterations and is removed from the neighbourhood of the affected part. The deposit, examined with the naked eye, is found to resemble plaster of Paris; examined under the microscope, it is seen to consist of minute crystalline needles of sodium urate, or bi-urate, often collected into stellate tufts and bundles. As already stated, the deposit is both intra-cellular and interstitial, that is, it

occupies the interior of the cells, and the interstices between them. When layers of cartilage thus affected are soaked for some hours in warm water, the deposit becomes completely dissolved, and the cartilage is almost or quite normal in structure. Wherever the deposit is found, the microscopic appearances are similar to those presented by the affected cartilage. If acetic acid be added, rhomboidal crystals of uric acid will appear. The *tophi*, or chalk-stones, often seen in the neighbourhood of the joints, consist of sodium bi-urate, with a little urate and phosphate of calcium. They differ entirely from the bony nodosities of chronic articular rheumatism. Their most favourite seats are the joints of the hands and feet and external ear. After some time, the skin covering them often becomes thin and brittle, and particles of chalk-like matter are found to cover the surface. In some cases the chalk-stones set up considerable irritation, and abscesses form, which, on bursting, leave ulcers. The discharges from such abscesses are milky and paste-like, and consist of sodium bi-urate, pus, and *débris*. The ulcers are indolent; the granulations are spongy, scanty, and yellowish; and the bases and edges are studded with particles of mortar-like

material. Such ulcers often remain without change for long periods, and are apt to re-open after cicatrisation. Another form of ulceration, due to the breaking-down of eczematous eruptions, is not unfrequent in gouty subjects. These ulcers, which are surrounded by eczematous vesicles, are apt to become very troublesome.

In order to complete this sketch of the morbid anatomy of gout, allusion must be made to the various lesions of internal organs, due to the existence of the gouty diathesis. Atheroma of the aorta is not unfrequent in chronic cases, and sodium urate has been found in the coats of the vessel. Cardiac hypertrophy, dilatation, fatty degeneration, valvular lesions and other evidences of chronic endocarditis are frequently found in advanced cases, and it is said that uric acid has been discovered in the morbid deposits on the endocardium. Pericardial adhesions and thickenings would appear to be sometimes associated with the gouty diathesis. With regard to the lungs, sodium urate has been found in the bronchial walls, and uric acid has been detected in the expectoration in a case of gouty bronchitis.

It is, however, in the kidneys of gouty subjects

that the most prominent and characteristic lesions are often discoverable, and a close study of these is especially important, as regards not only the etiology, but also the prognosis and treatment of the disease. Gouty nephritis, the so-called *gouty kidney*, is often found in severe and chronic cases, but a less serious affection not unfrequently complicates the slighter manifestations and earlier stages of the disease, and the lesions found in the kidneys may therefore be divided into two classes.

In the first class, the kidneys present deposits of uric acid and sodium urate scattered throughout their substance. These deposits are found in the cortical portion, in the pyramids, and in the papillæ, and likewise in the calyces and pelvis. The substance of the kidney is hyperæmic, and otherwise resembles in appearance that of the first stage of chronic interstitial nephritis. The changes may be regarded as the results of gouty inflammation, affecting not only the uriniferous tubules, but also the fibrous structures of the organ, and accompanied by the deposits of urates. Sir A. Garrod's view that the fibrous structures of the kidney are sometimes involved before the joints, is supported by those instances in which severe pain in

the back precedes and is relieved by the appearance of swelling in the great toe. The occurrence of temporary albuminuria is doubtless sometimes due to imperfect metamorphosis of albuminous substances by the liver, and perhaps to transient gouty inflammation of the kidneys. If, however, the albumen is permanently present, changes of a serious nature must have set in.

This question of albuminuria in connection with gout will be especially discussed in a subsequent chapter.

In the second class of cases, the kidneys present not only the infarctions above described, but in addition marked alterations of structure corresponding to those of chronic desquamative and interstitial nephritis. The infarctions appear as whitish streaks in the medullary substance, and the papillæ present little whitish points. The deposits are formed in and obstruct the uriniferous tubules, and are also found in the interstitial tissue. Under the microscope these infarctions are seen to consist of crystalline needles of sodium bi-urate, and they are chemically identical with the substance found in chalk-stones, and in the incrustations covering the articular cartilages. The structure of the kidney, moreover, presents marked

alterations. According to Charcot, there is at first a tubular nephritis, of two different degrees. In the first, the size of the kidney is unchanged, but the cortical substance is thickened and yellowish in colour. The Malpighian bodies are injected, and the uriniferous tubules are filled with epithelial cells, distended, opaque, and full of fatty or albuminoid granules. In the second stage there is atrophy of the cortical substance, and a granular condition of the kidney generally.

The alterations above described affect principally the uriniferous tubules, but in the gouty kidney, properly so called, there are likewise interstitial changes of a cirrhotic character. Thus there is thickening of the intertubular connective tissue and proliferation of the nuclei; the kidney becomes harder and diminished in weight and volume. The capsule is thickened and opaque, the surface is wrinkled and nodulated, and the cortical substance much reduced in quantity; in some cases the pyramids almost reach the surface of the organ. The infarctions of sodium urate appear as whitish streaks in the pyramidal portion. In some cases of this character the kidney has been found to weigh only two-and-a-half ounces.

Of the renal lesions just described the deposits of sodium urate may be designated as altogether peculiar to gout; for they are seldom, if ever, found in connection with any other disease. The other appearances are often found in that form of Bright's disease which is termed "cirrhosis" of the kidney. The co-existence of the urate infarctions with the contracted kidney frequently occurs in advanced cases of gout, and indicates a very close relation between the two diseases. There is also strong evidence in favour of the view that the kidneys are often affected at a very early period, even after a few moderate attacks have occurred. A granular condition of the epithelium, with excess of oil globules, and white streaks in the pyramids, constitute the earliest changes, while crystals of sodium urate occupy the intertubular fibrous structure and the tubules. The changes may co-exist with a perfectly natural size and weight of the kidneys, and with such a condition of their structure as appears normal to the naked eye. This fact has an important bearing upon the etiology of gout, and upon the part which renal disease takes in intensifying and perpetuating its manifestations.

Whether the kidneys simply excrete or really secrete

uric acid, disease of their tubular structure must affect the amount of this substance eliminated from the body, just as occurs in the case of urea.

In advanced stages the tubes become denuded of epithelium and are shrivelled and wasted. The Malpighian bodies are brought closer to each other and appear to have increased in number. The coats of the vessels, especially of the afferent arteries of the tufts, are thickened or hypertrophied, and the vessels themselves become tortuous. All these changes still further impede the excretion of the urinary constituents.

Sodium urate having been thus shown to be the *materies morbi* of gout, I shall, in the next chapter, discuss the existing theories with regard to the source and place of origin of uric acid.

# CHAPTER II.

## SOURCES AND PLACES OF ORIGIN OF URIC ACID IN THE SYSTEM.

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VARIATIONS IN QUANTITY OF URIC ACID UNDER VARIOUS CIRCUMSTANCES—DR. LEVISON'S CONCLUSIONS—IN DISEASE—INFLUENCE OF ACIDITY—MODE AND PLACES OF ORIGIN OF URIC ACID IN THE ECONOMY—TWO THEORIES: (1) FORMATION IN THE SYSTEM, ESPECIALLY IN THE LIVER; (2) FORMATION IN THE KIDNEYS ALONE—EXPERIMENTS ON BIRDS AND SNAKES—CONCLUSIONS TO BE ARRIVED AT—DR. PARKES' VIEWS—DR. LATHAM'S THEORY OF GOUT—GLYCOCINE THE SOURCE OF URIC ACID—RELATION OF URIC ACID TO GOUT—TWO THEORIES: (1) RETENTION OF URIC ACID IN THE BLOOD AS A RESULT OF DEFECTIVE ELIMINATION BY THE KIDNEYS; (2) URIC ACID INCREASED AS A RESULT OF SUB-OXIDATION OR OTHERWISE—DR. HAIG'S VIEWS—CONSIDERATION OF FIRST THEORY—OBJECTIONS TO IT.

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THE account given in the previous chapter of the pathological appearances observed in gout clearly shows that the deposition of sodium bi-urate in the cartilaginous structures of the joints is the fundamental *fact* upon which all theories as to the nature of the disease must be founded. The demonstration of an excess of uric acid in the blood constituted an important step towards the discovery of the cause of the

disease; but the sources and origin of uric acid in the system, even under normal circumstances, are still to be reckoned among the obscure points of physiology. It is therefore difficult to construct a complete and satisfactory theory which will explain the excessive production of this substance in disease. Before, however, entering upon the question of excessive production, it seems desirable first to discuss the various opinions now in vogue concerning the sources and places of origin of uric acid in the human body.

Under ordinary circumstances a healthy man excretes from 8 to 12 grains of uric acid ($C_5H_4N_4O_3$) *per diem*. According to the recent researches of MM. Yvon and Berlioz, the quantity eliminated is almost exactly the same for the two sexes. It exists in the urine in the form of acid urates of sodium and potassium. Sir W. Roberts states that quadriurates "are specially the physiological combinations of uric acid. They exist normally in the urine, and probably also in the blood." On the other hand, "free uric acid is not known physiologically, neither in the body nor in the urine." The quantity of uric acid rises and falls with that of the urea; the average proportion between the two substances being, accord-

ing to Dr. Haig, 1 to 33. Under conditions of abstinence from food, and when only non-nitrogenous substances are taken, the quantity excreted may fall as low as two or three grains; on the other hand, with a full animal diet it may rise as high as 30 grains. It is, however, a matter of fact that an excessive amount of uric acid is sometimes regularly excreted by pale and impoverished persons living on a diet poor in quality and not over-abundant. Under such circumstances the excess may be supposed to be due to imperfect metabolism. As a general rule, the variations in quantity are independent of differences in age, sex, height, weight, or temperature. In the colder months of the year, uric acid tends to accumulate in the body, and the excretion is lessened; in spring and summer, the excretion exceeds the formation, and some of the winter accumulations are removed. Moderate exercise appears to cause a slight diminution in the quantity; prolonged or violent exertion, on the other hand, produces an opposite effect. A decided diminution has been observed after copious draughts of water, and the quantity is said to be diminished after the use of alcohol, and, according to some authorities, after the administration of quinine

in full doses. Dr. Haig states that after a dose of quinine (six grains in 24 hours) the excretion of uric acid is at first diminished, but that later on an opposite condition obtains, and that the "plus excretion outweighs the minus excretion of the first stage." Iodide of potassium, chloride of sodium and caffein cause more or less diminution, though the statements with regard to salt are somewhat conflicting. The inhalation of oxygen is followed by rapid diminution of the uric acid, or even by its total disappearance from the urine. The administration of the carbonates of sodium and lithium has been thought to exercise a very decided influence upon the excretion of uric acid, inasmuch as urinary deposits composed of this substance rapidly disappear during a course of these drugs. It is, however, doubtful whether the uric acid is really *diminished in quantity*, and it would seem more probable that the disappearance of the sediment when alkaline carbonates are taken is due to the conversion of the acid phosphate of sodium into a neutral salt, and a consequently *diminished precipitation* of the uric acid. Besides this, some of the uric acid would unite with the alkalies, and form urates which are far more soluble than uric acid. Acids generally diminish the

excretion, but salicylic acid is said to be an exception to this rule. The salicyluric acid which it is supposed to form is much more soluble in water than uric acid. The use of mineral waters, containing the sulphate and carbonate of sodium, with a little chloride of sodium, is followed by a decided diminution of uric acid in the urine.

Dr. F. Levison* draws the following conclusions from an extensive series of experiments and observations :—

1. Uric acid is formed in the system as a result of the disintegration of the organised albuminous tissue-elements, and especially of the nuclein (a constituent of the leucocytes).

2. The excretion of uric acid is either increased or diminished by certain factors (*e.g.*, diseases, drugs, poisons, etc.) according as they accelerate or retard the disintegration of the cellular elements of the body, and especially of the leucocytes.

3. A supply of nutrient material, and especially of meat, causes a temporary leucocytosis, which is probably due to the nuclein contained in the food.

* "Die Harnsäurediathese," 1893, p. 25.

4. The quantity of uric acid excreted in 24 hours is not decidedly affected by the food supplied. A difference, however, exists between the effect of animal and that of vegetable food. The former, which is more easily digestible, causes leucocytosis and the formation of uric acid much more rapidly than the latter.

The known variations in the quantity of uric acid excreted in cases of disease are as follows :—

In febrile states of the system, *e.g.*, in traumatic fever, typhoid, small-pox, and acute rheumatism, the quantity of uric acid rises and falls with that of the urea. It is absolutely increased in those cases in which the elevation of temperature is associated with impediments to respiration, as occurs when such complications as pneumonia, capillary bronchitis, pericarditis, or pleuritic exudations are present. In cases of disease in which the action of the diaphragm is impeded from any cause, as in abdominal tumours, ascites, etc., there is an increased amount of uric acid in the urine. It would, therefore, appear that an increased excretion of uric acid generally occurs whenever there is *decreased energy* of the processes of *oxidation*.

In chronic gout, with deposits of urates in and about the joints, there is, as we have seen, a much diminished excretion of uric acid by the kidneys. In some forms of chronic splenic tumour, a similar condition is observed; but in others, and notably in cases of leucocythæmia, the quantity is often enormously increased. In one reported case, the daily excretion amounted to sixty grains. It may be that this increase is due to increased production in the diseased spleen, or to diminished oxidation in the system.

In anæmia and chlorosis, the uric acid is reduced in quantity, unless there be dyspnœa, in which case an increase is observed. In chronic affections of the respiratory and circulatory organs, accompanied by difficulty of breathing, and a consequent diminution in the supply of oxygen, the uric acid is increased in quantity. In carbonic oxide poisoning the proportion of uric acid to urea has been observed to be as high as 1 to 27.

In chronic affections of the kidneys, diabetes, and polyuria, the uric acid is generally diminished in quantity. In diseases of the liver there are considerable variations; in cirrhosis the quantity of sodium urate excreted is sometimes very high. Also in acute

congestive hepatic affections the quantity is much increased, but according to Dr. G. Harley * it is diminished in fatal permanent jaundice. In various forms of indigestion, and in cases of diminished cutaneous activity, sediments of urates are of common occurrence in the urine on cooling, but their precipitation must not be regarded as necessarily indicative of the presence of an unusual amount of uric acid. There is, moreover, as pointed out by Dr. Bence Jones, no relation "between the *acidity* of the urine and the *absolute amount* of uric acid which it may contain; for in the urine which is most acid, and which deposits the largest uric acid sediment, very little uric acid may really exist; whilst that which contains most uric acid may hold it in perfect solution, and may have but a feeble acid reaction." The presence of some other acid is doubtless the main cause of the deposit of uric acid sediments, for the addition of any acid to the healthy secretion passed soon after food is always sufficient to produce it. The temperature of the fluid also influences the precipitation; if this be high a larger amount of uric acid is

* "Diseases of the Liver," p. 761.

held in solution than under opposite conditions. Concentration of the liquid, on the other hand, favours the deposition of the uric acid sediment, for it augments the proportion of the urate to the water, and intensifies the acid reaction. "Thus the uric acid sediment may be regarded as dependent upon three concurrent conditions, (1) decrease of temperature, (2) increased proportion of uric acid compound to the water, positively or relatively, (3) increased acidity of the urine."* It is, therefore, evident that a copious precipitate either of uric acid or urates is no real indication of the quantity of uric acid actually present, which can be determined only by analysis.

Dr. Pfeiffer, of Wiesbaden, and Dr. Arnold Schetelig, of Homburg, have recently proposed a method of diagnosing gout, founded on the quantity of free crystalline uric acid present in the urine of patients exhibiting symptoms of the gouty diathesis. Uric acid in such patients is especially liable to precipitation, and if the urine be allowed to pass through a filter charged with uric acid—the "lithic filter"—the result will be that the free uric acid present will be retained,

* See Carpenter's "Principles of Human Physiology" ninth edition, p. 476

while the urates will pass through unaffected. It would seem that this uric acid is in the free, uncombined state, and the experiment tends to explain the peculiarly noxious effects even of small quantities of uric acid in gouty subjects, due to its remarkable proneness to become precipitated. Healthy individuals may excrete much larger quantities of uric acid without any bad results, inasmuch as there is far less liability to precipitation. Many experiments will, of course, be necessary before the value of this method of diagnosis can be precisely estimated. A description of the *modus operandi* will be found in the "Transactions of the Wiesbaden Congress for Clinical Medicine," 1889, and in the "Transactions of the Medico-Chirurgical Society of London," May, 1889.

Having thus described the principal variations occurring in the quantity of uric acid eliminated, the next subjects for consideration are the mode and place of origin in the human body of this important constituent of the urine.

Uric acid is one of the results of the metabolic processes which are constantly going on during life. Of the four principal elements, carbon, hydrogen, oxygen, and nitrogen, of which the body is composed,

and which constitute the bulk of the excrementitious materials, the nitrogen is eliminated almost exclusively by the kidneys, in the forms of urea and uric acid. These two substances differ in one very important respect, viz., as regards their solubility, urea being very soluble in water and in the fluids of the body; uric acid, on the other hand, requiring for its solution about eight thousand times its weight of distilled water at the temperature of the blood. Uric acid unites with various bases, and the salts thus formed are more soluble than the acid itself. They are, however, much less soluble than many other substances, and they and uric acid readily crystallise out from their solutions. It is owing to their insolubility and their proneness to reassume the solid form, that uric acid and its salts, though existing in such small amounts in the urinary excretion, so frequently give rise to disease. The fact that the urine of birds and of reptiles is composed almost entirely of uric acid is one of considerable interest as regards the relations of this substance to urea.

Two principal theories have been advanced with regard to the *place of origin* of uric acid in the animal economy. According to the first of these, uric acid is

formed in the system in general, during the changes which are constantly going on. Some authorities believe that it is produced mainly in the spleen, liver, and lymphatic glands; others think that the connective tissue is the principal seat of origin. It is certainly found in considerable quantities in the liver and spleen, but only slight traces are discoverable in the muscles. After its formation it passes into the blood, and is rapidly eliminated by the kidneys. According to this view, the kidneys merely serve as a filter through which the uric acid passes.*

In his recently published "Introduction to Human Physiology," Dr. Waller thus summarises the opinions now generally held as to the formation and excretion of uric acid. The liver forms not only bile-products, but also urinary products (urea, hippuric acid, uric acid), which are carried away in the blood and separated by the kidneys. The association between the liver and kidney as excretory organs may be characterised as *formation* by the liver and *separation* by the kidney. This relation, however, between the two organs must

* See Sir A. Garrod's Lumleian Lectures on "Uric Acid and its relation to Renal Calculi and Gravel," *Medical Times and Gazette*, 1883, vol. i.

not be regarded as absolute, or as implying a strict division of labour. Urea and hippuric acid are not formed from the liver exclusively; other organs, the kidney in particular, submitted to artificial circulation are capable of forming these substances. "The chief evident function of the kidney is the separation of formed materials; but it certainly also contributes to the actual formation of the materials that it separates." These views, thus concisely expressed by an eminent physiologist, are in accord with those which I have entertained for some years.

According to the second theory, uric acid is produced exclusively in the kidneys, and does not exist preformed in the blood. The epithelial cells of the uriniferous tubules are credited with the power of selecting from the blood the necessary materials, and of converting them into uric acid. The fact that uric acid has been repeatedly discovered in the blood appears to militate against this theory; but its presence is explained by assuming that it is absorbed from the cells of the kidney.

In reference to this important point, it may be useful to refer to some experiments in which the ureters of certain birds were occluded by means of

ligatures. As is well known, the urine of birds consists to a very great extent of uric acid, but this substance has not hitherto been detected in the blood of these animals. After the ligature of the ureters, incrustations of urates were found throughout the kidney (the Malpighian corpuscles alone excepted), on all the serous membranes, on the mucous membrane of the tongue, œsophagus and bowels, in the spleen, liver, gall-bladder, lungs, joints, muscles, bones and lymphatic vessels; only the brain and the blood remained free. The same results were obtained after ligature of the ureters in snakes; but after removal of the kidneys in these creatures deposits of urates were found only upon the edges of the wound, and in the places which the kidneys originally occupied. Inasmuch as when the kidneys were suffered to remain, and the ureters tied, copious deposits were found, but when these organs were removed, the deposits were almost absent, it might be inferred that uric acid is produced, in these animals at least, exclusively by the kidneys.

Several objections may, however, be raised against this interpretation of these experiments. If uric acid were formed in the kidneys alone we should not

expect to find it in the cicatrices left after the removal of these organs. Moreover, in the experiments in which the ureters were tied, with a copious deposit of urates in many organs and tissues as the result, we should expect to find uric acid in the blood, if this substance were formed in the kidneys, and carried by the circulation to the parts in which the deposits occur. It is well known that the tubuli uriniferi of serpents generally contain large deposits of urates, and it is quite possible that the incrustations found in various organs after ligature of the ureters may have resulted from the absorption of the deposits originally contained in the kidneys. Moreover, the discovery of urates in so many organs while the blood was free from them would seem to prove that the uric acid was actually formed in the organ in which it was found.

Other arguments might be adduced to show that the formation of uric acid takes place throughout the body. It may be that a minute quantity is produced in the kidneys as a result of the active changes going on in these organs; but the single fact that uric acid can be discovered in the connective tissue and in various organs in a state of health is conclusive evidence that its production takes place throughout

the body generally. A similar source of origin may be claimed for urea, which is most probably formed in all the glandular cells of the body, but especially in the liver (see page 54). According to Dr. Parkes,* in cases of hepatitis and hepatic abscess, when suppuration was excessive, the urea was found to be lessened in a degree proportionate to the extent to which the secreting structure was destroyed by the abscess. When the liver was not suppurating, but actively congested and enlarged, the amount of urea and uric acid seemed to be increased. With regard to the relations between these two substances, Dr. Parkes' opinion was that uric acid is not an anterior stage of urea, but has an independent origin of its own in some cells which are especially endowed with the power to form it. From his observations of several cases of enlarged spleen, attended by a nearly fourfold excretion of uric acid, he was led to suggest that the spleen may perhaps produce more uric acid than urea. Dr. Waller thus describes the transformation of *proteid* into *urea*. Proteid during digestion is converted into peptone, which is absorbed

* *Lancet*, 1871, vol. i., p. 467.

and re-transformed into the proteids of plasma and lymph, the *free circulating or coasting proteid.* Of this latter, only a small proportion is actually taken into chemical combination in protoplasm as *fixed* or *organ proteid*, the greater part being acted upon and used by living protoplasm without being integrated by it to make part of its own substance. The free proteid thus used up gives rise to urea, as does also the small proportion of fixed proteid which becomes disintegrated and has to be replaced. If excess of proteid be swallowed, it may in the intestine itself be converted into leucin and tyrosin, which, being absorbed into the portal system, may be at once converted into urea by the agency of the liver. Dr. G. Harley* thinks that urea is not a special product of the liver, but the ultimate product of all tissue disintegration. He admits, however, that in acute yellow atrophy of the liver, the amount of urea eliminated diminishes in proportion to the destruction of the liver-tissue. According to some experiments on fowls made a few years ago by Mach† the liver is not a special seat of the formation of uric acid in

* "Diseases of the Liver," p. 761.
† "Archiv. für exper. Pathol. und Pharm.," 1888, p. 389.

those animals. It was found that neither the formation nor excretion of uric acid was interfered with after ligatures had been placed on the hepatic vessels. On the other hand, Dr. Waller quotes Minkowski to the effect that after excision of the liver in geese, uric acid ceases to be excreted.

A theory recently advanced by Dr. P. W. Latham* with regard to the formation of uric acid, is thought by him to meet several difficulties connected with other views. Dr. Latham suggests that urea is formed from the glycocine (glycine) of the bile. After this fluid has served its purpose in digestion, the glycocine and taurine are returned into the blood, and are carried by the portal vein into the liver. In this organ these substances, together with leucine and tyrosine, are converted into urea; but if from any cause the metabolism of glycocine is interrupted while taurine and leucine, etc., still undergo the normal changes, the liver will contain both urea and glycocine. Dr. Latham further assumes that the conjugation of these substances by the gland would result in the production of certain compounds, one of which on arriving at the

* "On the formation of uric acid in Animals and its relation to Gout and Gravel."

kidneys is, in these organs, conjugated with urea and is excreted as ammonium urate.

This substance is very slightly soluble, one part requiring as much as 2,400 parts of water at 100° Fahr. for solution. In consequence of this property a certain portion may escape excretion and remain in the blood—"overflow, as it were, and so pass on into the circulation." In this fluid the ammonium salt meeting with the soda would be converted into sodium urate, the form in which it is deposited in and around gouty joints. Dr. Latham concludes that the appearance of uric acid in the secretion is the result of the imperfect metabolism of glycocine into urea, whether that glycocine be derived from the bile poured out into the duodenum or formed elsewhere in the body. That it is from the bile is made somewhat probable from the fact that in the carnivora, whose urine contains little or no uric acid, the bile contains no glycocholic, but only taurocholic acid, and therefore no glycocine.

In his Croonian lectures for 1886 Dr. Latham has still further developed this theory, and described very minutely the chemical changes which may be assumed to take place in the conversion of glycocine into uric

acid. Comparing gout and gravel with diabetes, he thinks that the liver is the organ at fault in these disorders. In diabetes, there is inability to effect the metabolism of glucose, which then passes into the circulation; while in gout and gravel the imperfect metabolism of glycocine is the primary and essential defect. "Unchanged it passes from the alimentary canal or elsewhere into the liver; there, under the action of the gland, it is conjugated with urea, resulting from the metabolism of the other amido-bodies, leucine, etc., and is converted into hydantoin; it then passes on to the kidneys to be combined with other molecules of urea or biuret, forming ammonium urate, a portion of which overflows into the circulation, and is converted into sodium urate. If the liver should become sluggish, as is likely to happen in persons who take too much food and too little exercise, or if "the terminations of the nerves should, from excessive stimulus, become somewhat paralysed, and the gland in some measure like the submaxillary after the injection of atropine, the result would be imperfect performance of functions and the non-metabolism of glycocine."

According to this view the imperfect metabolism of

glycocine is the primary and essential defect in gout and gravel, and therefore functional disorder of the liver is considered to take a prominent share in the production of these disorders. Dr. Latham, however, thinks that some change in the nervous system is the most important factor in their etiology, such change being either hereditary or acquired, and being situated in the medulla oblongata, spinal cord, or both. I shall have occasion to refer again to this theory in a subsequent chapter. The adoption of Dr. Latham's view as to the production of urea involves the acceptance of another and, as I think, an untenable theory, viz., that the formation of uric acid takes place in the kidneys. Moreover, our knowledge of the metabolic processes which go on in the organism is far too imperfect to allow us to assume that they are always identical with changes which can be produced in the laboratory.

The question as to the relation of uric acid to gout will now be discussed, and I proceed to inquire whether the disease is due mainly to the retention of uric acid in the system, or whether its primary cause is an increased formation of this substance, as a result of suboxidation of the tissues or otherwise.

The theory of the retention of the uric acid has of late years been upheld by several observers, and notably by Sir A. Garrod.* Whichever theory be adopted with regard to the origin of the uric acid, it is assumed that in gout the excretory power of the kidneys is defective as regards this constituent, though not as regards the urea and other substances. As a result of this defective capacity of the kidneys, the uric acid accumulates in the blood, and the prodromal symptoms of gout are likely to become manifest. If the patient's habits of life are such as to cause an increased production of uric acid, an attack of gout will sooner or later take place, and the disease will then pursue its course with more or less regularity. According to this theory, a functional affection of the kidney is the true pathogenetic cause of gout. The experiments in which the ureters of fowls and geese were tied, and the operation was followed by deposits of sodium urate in the joints, kidneys, and other viscera, have been thought to support this view.

* "A Treatise on Gout and Rheumatic Gout," third edition, p. 280. Sir A. Garrod says: "The imperfection in the eliminating power of the kidneys sometimes appears to be the chief, if not the only cause of the impurity of the blood." Sir A. Garrod of course allows that increased formation of uric acid plays a certain part in the production of gout.

It is necessary again to refer to Dr. Haig's investigations and opinions. He cannot find any evidence that excess of uric acid in the blood is ever due to increased formation. For every 33 grains of urea that are formed in the body, one grain of uric acid is also regularly and punctually formed, but no more. The 33 grains of urea are "excreted with practically absolute certainty," but the insoluble uric acid is very apt to lag behind, and to be retained in various tissues and organs, which are less alkaline than the blood, and therefore less able to hold it in solution.* The blood never becomes loaded with uric acid, except as the result of previous imperfect excretion. He adopts Sir A. Garrod's view that "the antecedent elements of uric acid come probably from the liver to the kidney, and are there formed into urate of ammonium."

There are, however, many objections to this theory. Gouty deposits do not necessarily take place in connection with those diseases of the urinary organs which are attended with a much diminished secretion of urine. Moreover, gout often occurs in individuals

* "Uric Acid as a Factor in the Causation of Disease," p. 43.

whose kidneys are to all appearances healthy, but whose blood can be shown to contain an excess of uric acid. If the urine be examined in the early stages of gout, there is, as a general rule, no evidence of any renal affection. It is quite true that, on post-mortem examination, the kidneys of gouty subjects are often found to contain uric acid infarctions and to present other morbid appearances, but these are the consequences and not the causes of the gouty dyscrasia. It may readily be conceived that the irritation, often repeated or almost continuous, which deposits of urates in the uriniferous tubules must sooner or later set up, will give rise to serious changes in the renal tissue, and the deposits which occur in the interstitial tissue of the kidneys may be regarded as the result of an attack of gout localised in these organs. Persons who have never had any attacks of gout, but whose urine often contains a deposit of uric acid, are especially prone to suffer from inflammation of the kidneys, and the relationship between gout and uric acid deposits is based upon the fact that the uric acid dyscrasia is common to both conditions.

Uric acid deposits and gout are frequently associated in the same subjects, but cases sometimes

occur in which there are severe paroxysms of gout, but no marked deposits of uric acid in the urine. Under such circumstances neither albumen nor casts are likely to occur. Such patients are, however, very liable to renal inflammation, and when this occurs there will be an increase of uric acid in the blood and a diminished amount in the urine. If the mere occlusion of the tubules could give rise to gout, or if the excess of uric acid in the tissues of gouty persons were the consequence of its retention in the kidneys, the disease would be of far more frequent occurrence, and even infants would be liable to gouty deposits, inasmuch as Virchow has shown that the kidneys of new-born children frequently contain uric acid infarctions. In addition to this, it is well known that many persons suffer more or less from gravel during their whole life, but are quite free from attacks of gout. Some members of a gouty family exhibit all the symptoms of the disease; others suffer only from gravel. It can easily be understood that an affection of the kidneys which is accompanied by a lessened excretion of urinary constituents will tend to increase the frequency and severity of the gouty attacks. So long as the kidneys remain sound, a rapid excretion prevents undue accumulation.

The experiments above referred to in which ligature of the ureters in geese and chickens was followed by deposits of urates in and upon various organs will scarcely help us to explain the phenomena of gout. If the human organism produced as much uric acid as that of birds, and if the flow of urine through the ureters were absolutely prevented so that no more could possibly be removed by the kidneys, it is quite conceivable that the uric acid would be deposited in and upon all organs of the body adapted for its retention. There is, however, a very decided difference between such deposits and those of gout occurring in an individual whose urine is free to escape.

The theory that in gout the uric acid excreting function of the kidney is defective, rests upon a pure hypothesis, and could be accepted only if a more suitable theory could not be discovered. Even those who support it admit that it accounts only for the prodromal symptoms of gout, and they allow that an increased production of uric acid is necessary for the full development of the disease. Such increased production will form the next point for consideration.

# CHAPTER III.

## THEORIES AS TO THE NATURE OF GOUT.

Excessive Production of Uric Acid in Gout—Circumstances influencing the Excess—Formation of Urea and Uric Acid by the Liver—Functions of the Liver—Dr. Paton's Researches on the Relationship of the Formation of Urea and Uric Acid to Secretion of Bile—Lithæmia due to Hepatic Derangements — Symptoms of the Uric Acid Diathesis—Drs. Graves and Leon Williams on Gouty Disorder of Teeth—Occurrence of Temporary Albuminuria in Gouty Dyspepsia—Eczema as a Symptom of the Gouty Diathesis—Sir W. Roberts' Views as to the Mode of Action of Uric Acid—Neurotic Theories of Gout—Views of Sir Dyce Duckworth, Dr. Meldon, Dr. E. Liveing, Sir J. Paget, Dr. Ord, and Dr. Latham—The Author's Views.

TOWARDS the close of the preceding chapter I endeavoured to show that defective elimination of uric acid is not sufficient to account for its undue accumulation in the blood in cases of gout. I now proceed to discuss another theory, viz., that of excessive production of uric acid in the system, a condition which is liable to occur under the following circumstances :—

In the first place, albuminous constituents may be supplied in excess, and the surplus fails to become properly oxidized. In the second, the oxidizing pro-

cesses are imperfectly performed; though the supply of those constituents may be no more than is fairly proportioned to the normal wants of the organism. There is also a third class, in which not only are the oxidizing processes far below the normal standard, but the supply is manifestly in excess of the requirements. Under all these conditions the blood is liable to become surcharged with imperfectly oxidized constituents, of which uric acid is, for our present purposes, the most important.

In the healthy organism by far the larger part of the nitrogenous excreta appears as urea, which is more highly oxidized than uric acid. Whether the entire amount of urea which is eliminated from the system results from the oxidation of this latter substance—that is, whether uric acid forms a necessary stage in the production of the urea—is a question which must be answered in the negative. It is possible that uric acid may be only one of several substances, out of which urea is formed; and in a healthy organism its production may be the result of changes slightly divergent from those concerned in the formation of urea. It has been already stated that in all probability several tissues of the body participate in the produc-

tion of these substances, but there is strong evidence in favour of the view that the liver is the principal seat of their origin.

Up to within quite recent times the secretion of bile was thought to constitute the principal, if not the only function of the liver; though it would appear that the earliest writers on medicine had far more extended views as to the work performed by this organ. It now seems clear that these old views deserve to be revived, and that the liver must be credited with the discharge of at least *three* functions, viz.: (1) the secretion of bile, (2) the formation of glycogen, and (3) the destruction of albuminous matters derived from the food and tissues, and the formation of urea and uric acid. It is with this third function that an endeavour to trace the origin of gout is closely concerned, and it is to the writings of the late Dr. Murchison that the profession is especially indebted for a clear exposition of the present state of knowledge on this subject,* and for the inferences to be deduced therefrom. His observations show that the liver is largely concerned in the formation of the nitrogenous matters which are

* Murchison, "Functional Derangements of the Liver," second edition, 1879.

eliminated by the kidneys. Some evidence in favour of this theory has been adduced in preceding pages, but there are other facts which deserve attention. Deposits of uric acid and urates, and an imperfect formation of urea, are frequent signs of functional as well as of organic affections of the liver; while there is experimental evidence of the existence and formation of urea in this organ.

Another fact, referred to by Dr. Murchison, has an important bearing on the subject of the hepatic functions. It appears from Bernard's experiments that the temperature of the healthy liver reaches 104°, or even 106°. In dogs, the temperature of the blood in the hepatic veins is much higher than that of the portal vein, and the temperature of the upper part of the vena cava is higher than that of any other part of the body. The active chemical changes going on in the liver are the probable cause of this elevation of temperature, and this view is further supported by the fact that in diseases of the liver the temperature is often lower than normal, and that after the common duct has been tied in animals there is no longer any difference in temperature between the blood of the portal and that of the hepatic veins.

In some very interesting lectures* on the "Relationship of the Formation of Urea and Uric Acid to the Secretion of Bile," Dr. Noel Paton has shown that the liver is the part of the animal economy in which urea is principally formed. He has demonstrated, by conclusive experiments, that stimulation of the flow of bile by means of drugs (salicylate of sodium, benzoate of sodium, colchicum, perchloride of mercury, euonymin) is accompanied by an increased production of urea, and not merely by an increased elimination.

If this view of the function of the liver be correct, it is easy to understand how the condition termed *lithæmia*, in which there is an excessive amount of uric acid in the blood, may often depend upon derangement of the liver, and therefore that the gouty dyscrasia may have a similar causation. If there be an imperfect transformation of albuminous matters, with the production of uric acid instead of urea as a consequence, we have at least *one* of the factors necessary for the development of gout. The most common cause of such imperfect transformation is an

* *British Med. Journal*, 1886, vol. i., pp. 377, 433; vol. ii., p. 207.

excess of supply, combined (as it very often is) with deficient action of the assimilating organs. It is well known that an occasional deposit of urates in the urine is a common result of over-indulgence at the table, and is of no serious significance. When, however, such a deposit is constantly or even frequently noticed, it is of more or less grave import. Patients are apt to think that the kidneys are "out of order," but in point of fact it is not these organs, but the liver, which is generally in fault; and this distinction is one of paramount importance as regards the treatment, whether curative or prophylactic, of such cases.

When these deposits are of constant occurrence, the uric acid diathesis may be said to have become established; and in the majority of cases other symptoms, perhaps noticed occasionally before, soon become troublesome. There is more or less dyspepsia, as evidenced by flatulence, distension, and feelings of uneasiness, or even severe pain, in the stomach and duodenum.* In addition to pain, there is often

* The signs of gastric disorder may be the principal feature of the condition. In a case recorded by Dr. Bence Jones, the patient, a gentleman, aged 40. had been liable to constant deposits of uric acid and urates in the urine. He afterwards suffered from attacks of violent intermittently spasmodic gastralgia, coming on from one to five hours after a late dinner. These lasted for about an hour and then passed off

nausea and either pyrosis or acid eructations; in many cases there is a bitter taste in the mouth, the tongue is dry and furred, the bowels are irregular and generally constipated, the liver is somewhat enlarged and tender on palpation, the skin sometimes has a slightly jaundiced hue. The appetite is irregular and capricious; the patient often exhibits a preference for highly spiced food and acids, which stimulate the torpid stomach. Palpitation of the heart and shortness of breath, aggravated by exertion, are often complained of, and a short dry cough is sometimes very troublesome.* Hæmorrhoids in various stages are not unfrequent, and when present in these cases are indicative of hepatic congestion. Symptoms of derangement of the nervous system are almost always superadded. The temper becomes irritable and the spirits are depressed. The patient feels uneasy and restless, or else there is lassitude, drowsi-

and were followed by deposits of lithic acid crystals in the urine. Under carefully regulated diet and the use of alkalies, the attacks entirely ceased. A case of a similar kind, occurring in my own practice, will be mentioned in a subsequent chapter.

* Dr. Woakes (on "Post-Nasal Catarrh") has described many of the symptoms of the uric acid diathesis as those of "the premonitory stage of catarrh," and has explained in a very lucid manner the decided predisposition towards taking cold evinced by many subjects of this diathesis.

ness (especially after food), headache, and inability for mental exertion. The sleep is broken and unrefreshing. Noises in the ears and vertigo are sometimes very troublesome and alarming. Hypochondriasis is not unfrequent, and in women hysterical symptoms may occur. In some cases frequent attacks of migraine, or of other forms of neuralgia, are prominent symptoms; in others, the small joints are painful from time to time, but there is no decided attack of gout. Transient attacks of conjunctivitis ("hot eyes," as described by Mr. Hutchinson) are common in many patients. Severe cramps in the legs and other parts, pains in the heels and painful sensations of burning and tingling in the feet are sometimes present and cause great annoyance.

Chronic catarrh of the fauces is occasionally connected with lithæmia, and is likely to prove very obstinate until suitable treatment is employed. The appearances consist in patches of congestion at the back and sides of the pharynx, and the patient complains of uneasiness or of some amount of pain on swallowing. This feeling may extend to both ears and to the adjacent parts of the neck, and the pain and discomfort are often much more severe than the

local appearances would lead one to expect. Periodicity is sometimes a prominent feature, and the symptoms are almost invariably aggravated by astringent and stimulating applications.

Another very troublesome but rare symptom of lithæmia was first described by Dr. Graves.* This consists in an insuperable desire on the part of the patient to grind his teeth; it originates in an uneasy sensation in the teeth themselves, and can be thus momentarily alleviated. The practice becomes habitual, and after some years the conformation of the teeth undergoes serious changes; these organs may, in fact, become ground down to the level of the gums. Gastric disorder increases the propensity. In Dr. Graves's cases, all occurring in persons of the gouty diathesis, no means could be discovered for the alleviation of the symptom. Loss of teeth, apparently sound, is somewhat frequent in lithæmic patients of middle life; and attacks of pain in several sound teeth at the same time, with peculiar sensations as if the teeth were starting from their sockets, have also been observed in the same class of persons.

* "Clinical Lectures on the Practice of Medicine," New Syd. Soc. Ed., vol. i., p. 534.

Cases of the kind referred to by Dr. Graves have not as yet come under my own observation. My friend, Dr. J. Leon Williams, D.D.S., has been kind enough to furnish me with the following account of his experience with regard to the teeth of gouty persons. "Recent investigations have shown the intimate relationship existing between gout and that oral disease, *pyorrhœa alveolaris,* which attacks the peridental membrane covering the roots of the teeth and lining the bony sockets in which they rest. Many various symptoms are presented, but the one unmistakable indication of true pyorrhœa is discharge of pus from the gums about the necks of the teeth. These pus-pockets are sometimes deep down the side of the root of the tooth, but their presence is easily detected by making pressure with the finger along the gum in a line with the axis of the tooth; on the upper jaw from above downward, and on the lower jaw from below upward, thus forcing the pus to the surface. In advanced stages of the disease, a probe can often be passed the entire length of the affected tooth, and a nodular, limy deposit can usually be detected on the tooth throughout the extent of this pus-channel. This deposit, which is quite distinct from

salivary calculus or tartar, yields, on analysis, uric acid, calcium urate and allied compounds."

The local treatment consists in carefully removing the uric-calcic deposits, syringing the pus-pockets with antiseptic solutions, followed by astringents (aromatic sulphuric acid and sulphate of copper), and germicides, such as oil of cassia, aristol or hydronaphthol.

It is obvious that the symptoms mentioned in the foregoing paragraphs are connected with some peculiar condition of the system at large. They are liable to periodical exacerbations, and they are invariably aggravated by errors of diet. A little beer, a glass or two of champagne and acid beverages in general will often cause twinges in the knuckles, burning sensations in the palms and soles, or even more decided manifestations in these subjects.

Cases presenting many of the above symptoms, variously combined, often come before me in practice, and I never fail to examine the urine, which, in not a few instances, I find to contain a distinct quantity of albumen, without tube-casts. In a succeeding chapter I shall endeavour to explain this appearance; for the

present it will be sufficient to say that I do not regard the albuminuria in these cases as necessarily indicative of pathological changes within the kidneys. The urine is likewise scanty, generally high-coloured, and abnormally acid; after standing, it deposits an abundance of urates and uric acid. Oxalate of lime is often present. Precipitation of urates sometimes takes place within the bladder, and in that case the urine is more or less turbid when passed.

In a majority of cases the attack of articular inflammation is preceded by many of these symptoms, and appears to follow them directly; while in some decidedly gouty persons no other signs of gout than those which characterise the uric acid diathesis occur at any period of life. It may be said that in these latter cases the acute phase is never reached. It is worthy of remark that some patients who suffer from aggravated dyspepsia, and from gout, find themselves very much better than usual, as regards the dyspeptic symptoms, for some little time before the acute attack.

There is one other symptom which I must not fail to mention. In many of my patients presenting the symptoms of gouty dyspepsia, I have noticed the

occurrence of eczema, which appeared to alternate with the gouty symptoms, inasmuch as it increased in severity as the latter were relieved by the treatment. The dyspeptic symptoms can be removed by very careful attention to diet and appropriate medicines, and, if due care be taken, they will not necessarily recur. The patient, however, is still liable to attacks of eczema, especially in the spring of the year, and the complaint, in the absence of further treatment, tends to become almost continuous. This symptom is especially troublesome when, as often happens, the parts about the anus are affected. Pruritus ani is commonly associated with eczema. Dr. Robinson, in his recent work on this latter complaint, states that he has "never examined any case of itching about the anus without discovering evidence of inflammatory action, and that in every case this action has produced manifestations which could only be classified as eczema." I shall have more to say on the connection between eczema and gout in a subsequent chapter.

In his "Croonian Lectures," 1892, already referred to, Sir W. Roberts discusses the mode in which uric acid produces its injurious effects. He comes to the conclusion that the latter are due to the mechanical

damage consequent on its precipitation as sodium biurate in the tissues ("uratosis"), and not to any poisonous properties inherent in uric acid circulating in the blood in a state of solution. This mechanical theory explains the incidents of regular gout in a manner which may be described as natural and complete; but at first sight, it appears to be inapplicable to the manifestations of irregular gout. Sir W. Roberts, however, suggests that these manifestations are really due to uratic deposition (as is the case with those in the joints), the crystals falling "in gentle sprinklings, sufficient perhaps to cause irritation, if the implicated tissue be a sensitive one, but not enough to cause downright inflammation." It is true that such slight precipitations have seldom, if ever, been actually found; but they are probably evanescent in character, and are often removed as soon as the blood recovers its solvent power. Prof. Ebstein's view is to the effect that neutral sodium urate is deposited in the affected tissues and produces necrosis. The portions thus destroyed act as irritants to the surrounding parts in which crystalline urates become deposited.

An attempt to explain the nature of gout would

obviously be imperfect without taking into consideration the evidence in favour of the *neurotic* origin of the disease. By some authorities gout is regarded as a disorder of the nervous system, that is, as a trophoneurosis—a view which has recently been advocated by Sir Dyce Duckworth.* This would appear to be a convenient place for an account of the arguments brought forward by him and other writers in support of this theory.

Sir Dyce Duckworth admits that it is impossible to resist the evidence in favour of the direct connection between the excess of uric acid in the blood and the manifestations of gout, and that whatever views be entertained as to the *whole* of the pathogeny of this disease, the *facts* discovered by Sir A. Garrod cannot be set aside. The question, however, is whether the theory of the excess of uric acid being the cause of gout is *sufficient* to account for all the phenomena. Cullen, as already mentioned, regarded gout as manifestly an affection of the nervous system, and asserted that gout was more indicative of nervous disorder than any other pyrexia. Stahl, however, was the first

* *British Medical Journal*, March 26th, 1881. See also "A Treatise on Gout," 1889, p. 44, etc.

to advocate the neurotic theory of gout, and Cullen adopted his views.

Diseases belonging to the neurotic type may be either primary or central, or may be secondary or induced, and it is alleged that gout presents not a few of the characteristics peculiar to the neuroses, *e.g.*, heredity, the periodicity of the attacks, and subjection of the disease to the law of alternation. Primary gout is regarded as a *diathetic* neurosis, but a difficulty arises in accounting for cases of gout occurring in the absence of any neurotic taint. It is assumed that, at least in some of these, a condition of lithæmia and hyperinosis is set up as a result of high living, etc., and that the consequent dyscrasia of the blood reacts upon the nervous centres; in other words, that a secondary affection of some nerve-centre occurs in consequence of the altered state of the blood, and thus the order and special phenomena of the gouty attack become developed. The fact that joint-affections are occasionally witnessed in the course of diseases of the cord and brain is also adduced as a strong argument in favour of this theory; and the gouty arthritis is supposed to be connected with these affections. In accordance with the suggestion which

was first made by Dr. Buzzard,* a trophic centre for joints is assumed to exist near the roots of the vagus in the medulla oblongata.

In addition to such features as heredity and periodicity, gout is considered to present many other analogies with disorders of the nervous system. The sudden supervention of the attacks; the preceding sensations of ease and comfort so often experienced; the time of the occurrence of the attacks and their paroxysmal character; the connection of the disease with other well-recognised neuroses; and the fact that the same causes are liable to excite attacks, have all been cited as evidences of the close relation which exists between gout and the nervous system. The remarkable connection between gout and diabetes is also adduced; it being a well-recognised fact that the two diseases sometimes alternate and that members of gouty families are prone to suffer from diabetes. It is also claimed that this theory of the origin of gout serves to explain the effect of colchicum—a drug which has a powerful action upon the nervous system.

* "Transactions of the Pathological Society," 1880, p. 208.

Dr. Meldon,* of Dublin, has long advocated what may be termed a neuro-humoral theory. He admits that uric acid and soda must exist in the blood before the disease can be produced, but is convinced that the presence of these substances is not the sole cause of gout. His view is that there must be *depression of the nervous system* to cause an attack of gout—such depression bringing about the union between uric acid and soda so as to form the urate of soda. According to this theory, nervous force when in a normal condition keeps these two substances separate and in a fluid state, and in a condition fit for elimination by the skin, kidneys, and bowels. When the nervous influence is lessened, the uric acid and soda unite in the tissues most removed from the blood and circulation. The irritation and inflammation excite the nervous system to increased energy, and the disease for the time is arrested. One objection to this theory is constituted by the fact that urate of sodium is far more soluble than uric acid, and therefore more easily eliminable by the kidneys.

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* "Gout, Rheumatism, and Rheumatic Gout," 1872; and *British Medical Journal*, vol. i., 1881, p. 466.

Dr. E. Liveing,* in his classical work on Megrim, is much inclined to assent to the view that gout in its various forms is the manifestation of a disorder which has its primary seat in the nervous system itself; and he lays great stress on the connection which may often be traced between gout and such disorders as "megrim, asthma, angina pectoris, gastralgic paroxysms, and certain forms of transient mental derangement." Sir James Paget expresses a somewhat more guarded opinion with regard to the part played by the nervous system in the causation of gout.† He says: "Disturbance in the nervous system in some form and part may be regarded as a factor in every case of gout. There are reasons enough for thinking that changes in the nervous centres determine the locality of each gouty process, while changes in the relations of the blood and tissues determine its methods and effects; and that thus we may explain the symmetries of disease in gout, sometimes bilateral, sometimes antero-posterior, and thus its metastases. But these changes are a

* "Megrim, Sick-headache, and some Allied Disorders: A Contribution to the Pathology of Nerve-Storms," 1873, p. 404.

† "Clinical Lectures and Essays," 1879, p. 382.

part of the pathology of gout which is not yet clinical."

According to another theory, proposed by Dr. Ord,* the part played by the nervous system in this disorder is held to be the propagation of gouty inflammation from part to part. It is also considered that there are evidences of the direct action of the nervous system in the production of the attacks. When the gouty diathesis exists any sudden excitement of the nervous system is capable of producing gouty inflammation in a violent form, and in several parts at once. As to the nature of gout, Dr. Ord's views are that the disease is a mode of decay of the whole system; that the deposit of urates is the result of local or general disintegration, the local inflammation not necessarily depending upon such deposit, but often set up by local exciting causes; that the local inflammation and degeneration tend to infect the rest of the system through the blood, and to set up similar actions elsewhere through reflex nervous influence. These views have been adopted in the main by Dr. Bristowe.† Dr. Wade's theory has been already mentioned.

* "St. Thomas's Hospital Reports," 1872.
† "The Theory and Practice of Medicine," fourth edition, p. 874.

A reference to the recently published views of Dr. P. W. Latham* will conclude this part of the subject. Dr. Latham thinks that some change in the nervous system is the most important factor in the etiology of gout; and that the change is localised in the medulla oblongata or spinal cord, or both, and that it may be either hereditary or acquired. He considers (1) that derangement of the hepatic functions leading to excessive formation of uric acid may be connected with some "weak spot" in a portion of the medulla oblongata involving some of the roots of the vagus; (2) that uric acid in the blood resulting from hepatic derangement might act upon any weak spot near the roots of the vagus and cause gastric troubles, asthma, cardiac irregularities, etc.; (3) that if in addition to some change in the medulla oblongata, those portions of the spinal cord which control the nutrition of the joints are affected, articular inflammation with deposition of sodium urate will become developed.

Many attempts have been made to explain the occurrence of gouty deposits in the joints and fibrous structures rather than in other tissues of the body.

* "On the Formation of Uric Acid in Animals," etc., 1884, p. 30.

Several hypotheses, advanced by various writers, have been already noticed. According to the so-called physical theory, the phenomena of gouty attacks are attributable to local influences and may be thus briefly described. Assuming an increased formation of uric acid, " or an accumulation in the textures (especially the fibrous) brought about either in connection with a tropho-neurosis, or, as some believe, by vital decay, some of the tissues of the body hold more uric acid than can be destroyed. In the more vascular parts, the excess is washed away by the more rapid current and appears in the blood, whilst in the least vascular, the cartilages of the joints, especially those more remote, the force of the blood-current is too feeble to ensure removal and consequently an attack of gouty inflammation ensues. With repeated attacks, the system gets more and more saturated with the accumulated uric acid, the larger joints become invaded and the straight tubules of the kidney blocked sooner or later with uratic deposits."* Sir W. Roberts lays stress upon the influence of synovial fluid upon the deposition of urates. This fluid is

* *Lancet*, 1888, vol. i, p. 283.

comparatively motionless and contains a relatively large percentage of sodium salts, the presence of which is distinctly favourable to uratic precipitation.

Such then are the theories now in vogue with regard to the pathology of gout. The fact that the blood in this disorder contains urate of sodium is common to them all, but it is evident that very different views are held as to the part played by this substance in the causation of the phenomena. I have given a somewhat lengthy account of the *nervous* theory of the disease, because it is advocated by many distinguished authorities at the present day, and bids fair to gain increased acceptance. It appears, however, to be open to several objections, and cannot, I think, be adopted in its entirety, especially in the form given to it by Dr. Latham.

It must be admitted that disorder of the nervous system is a prominent feature in many cases of gout; but it is surely going too far to assume that this disorder is the *primary cause* of the phenomena, the *fons et origo mali*. Disturbances of the nervous system are common enough in many disorders universally believed to be due to the presence of some morbific matter in the blood, and it is generally thought that

the poison exerts some peculiar action upon the nervous centres. Now, in gout we have a poisonous material circulating with the blood, and that the nervous system should be affected by it is at the least highly probable, judging from our experience of fevers, pyæmia, etc. Dr. Latham's ingenious hypothesis of a "weak spot" in the medulla oblongata is surely unnecessary for the explanation of lithæmia, and we know too little about a "trophic centre for the joints" to be able to trace gouty attacks to primary disorder of that portion of the nervous system. In the condition known as pyæmia we often meet with suppurative inflammation of joints far removed from the original wound. It is highly probable that the nervous system is deeply concerned in these diffused "metastatic" inflammations; but it is clearly impossible to assume that *antecedent* mischief or "weak spots" in trophic centres determine the incidents and course of the morbid phenomena.

My theory with regard to gout recognises the cause of the disorder in the presence of abnormal quantities of sodium urate in the blood. I am willing to admit that many of the symptoms are indicative of nerve disturbances, but these latter are due to the poisonous

action on the nervous centres of the materials formed in the body, and resulting from imperfect metabolism. The various neuroses which often affect gouty subjects probably depend upon the operation of the same cause. The hypothesis of antecedent nervous lesions is at least unwarranted.

A consideration of the statements contained in the foregoing paragraphs will, I think, justify the following propositions:—

1. Uric acid, in the form of sodium urate (or biurate) is the *materies morbi* of gout.
2. The deposit of sodium urate in the joints is the cause of the gouty inflammation.
3. This substance is produced in excess, as a result of the imperfect transformation of albuminous substances.
4. This imperfect transformation is for the most part due to functional disorder of the liver, or to excessive supply of nutritive materials, or, as often happens, to a combination of these causes.
5. So long as the excess of uric acid is eliminated by the kidneys, decided attacks of gout may be absent; but the symptoms above described as

pertaining to the uric acid diathesis are liable to be present.

6. The kidneys are apt to become *secondarily* affected, owing to the irritation set up by excess of uric acid and other products of defective metamorphosis, and by deposits of urates. Primary disorder of the kidney is not a necessary factor in the production of gout.
7. In the majority of cases of chronic gout, increased production of uric acid is associated with defective elimination by the kidneys.
8. The symptoms of nervous disorder in gout are due to the action of the *materia peccans* on the nerve-centres.

# CHAPTER IV.

## CAUSES OF GOUT.

PREDISPOSING CAUSES—HEREDITY—THE ACQUIRED FORM—GEOGRAPHICAL DISTRIBUTION—SEX AND AGE—CLIMATE AND SEASON—ERRORS IN DIET—EXCESS OF ALBUMINOUS FOOD—WANT OF EXERCISE—DR. CARPENTER ON DIET AS A CAUSE OF GOUT—OTHER ALIMENTARY SUBSTANCES—STARCH, SUGAR, AND FATS—ACIDITY—INFLUENCE OF FERMENTED LIQUORS—LEAD—MENTAL INFLUENCE—DEPRESSING AGENCIES—EXCITING CAUSES OF GOUTY PAROXYSMS.

HAVING discussed various theories as to the nature and *immediate* cause of gout, and stated my own views on these subjects, I proceed to consider what are usually termed the *predisposing* causes of the malady. While regarding gout as in very many cases the result of functional disorder of the liver, it cannot be denied that other factors of a more or less potent character often aid in the production of the disease. I shall therefore devote a few pages to a consideration of the most important of these causes, and also endeavour to trace the origin of the hepatic derangements.

Gout is referred to in the writings of Hippocrates, and there are many notices of the complaint in the works of various Roman authors, some of whom allude to its increased frequency in their experience.

The *hereditary transmissibility* of gout (or of the gouty diathesis) has long been recognised, and has been proved beyond all question; in this respect the complaint manifestly resembles other forms of perverted nutrition. Galen attributed the increasing spread of the disease in the Roman empire to the influence of heredity. Among 522 gouty persons, concerning whom Sir Charles Scudamore had collected information, 332 gave evidence of the disease in some direct or collateral ancestor. Hereditary predisposition can be traced in about 55 per cent. of all cases, though probably even this estimate is somewhat too low. While due weight must therefore be allowed to hereditary influence, it is, on the other hand, perfectly certain that gout can be *readily acquired*. This latter form generally appears later in life than the hereditary disease, symptoms of which are often developed at a very early period. In families in which a gouty taint exists, the disease is apt to show itself most frequently

and most severely in the younger children, inasmuch as the dyscrasia generally becomes intensified in the parents with advancing years. In some instances the gouty tendency skips over a generation; the son of a gouty father perhaps suffers only from indigestion, or exhibits marked obesity, but no definite gouty symptoms, whereas his offspring are severely affected by the disease. As a matter of course, the influence of hereditary predisposition may be greatly strengthened by the patient's habits of life.

With regard to *geographical distribution*, gout is said to be most common in England and in the southern provinces of Italy. Professor Cantani, of Naples, states that in his country the hereditary tendency has been handed down from the period of the Greek colonisation and the Roman Empire. Dr. Hirsch tells us that in the German Empire gout is comparatively frequent in Hamburg, Mecklenburg, Bremen, Göttingen, Dresden, and Wiesbaden; in Berlin it is somewhat rare. With regard to the United States, my own experience leads me to believe that gout is becoming somewhat frequent in that country. Speaking generally, *acute* gout has become less common during the last few decades, and this

change must be attributed to the greater prevalence of temperance. Evidences of a gouty taint are, however, still very common; many persons who would have suffered from the disease in its old-fashioned form had they adopted their ancestors' habits, are now troubled by dyspepsia, eczema, various forms of neuralgia, and other manifestations of lithæmia.

It is not surprising to find that *women* are much less liable to suffer from this disease than *men;* the proportion of females to males is somewhat less than 3 per cent. Chronic rheumatism, on the other hand, is more frequent among women, and thus differs from gout. The female descendants of gouty ancestors are especially prone to nervous symptoms, such as neuralgia. It is also worthy of note that in women the attacks of gout usually come on at a later period than in men, in most cases, indeed, not until after the menopause, unless the hereditary tendency is very strongly marked. Gout very rarely develops itself before the *age* of eighteen; first attacks are most common between the ages of thirty and forty. I have met with one case of unmistakable gout in a girl of 16, whose father and maternal uncle were martyrs to the complaint. It is doubtful whether any particular

temperament or constitution can be regarded as a predisposing cause of gout.

With regard to *climate* it cannot be said that this has any direct effect on the causation of gout. The complaint is almost entirely confined to the temperate regions of the earth, and natives of hot countries (those of southern Italy excepted) appear to be quite exempt from any of its manifestations. On the other hand, Europeans living in hot climates and indulging freely in animal food and fermented liquors are almost as prone to suffer as they would be at home. In the palmy days of the East India Company, when high living and copious potations were almost universal, gout was by no means an uncommon disease among the servants of the Company, but the climate had no real share in its production. Dr. Norman Chevers,* indeed, thought that, "with all its sanitary faults Calcutta is an earthly paradise for those cautiously-living people, whom gout threatens, but has not crippled." It is worthy of note, as showing the influence of season, that first attacks of gout are most common in spring and autumn, and that recur-

* *Medical Times*, August 30th, 1884, p. 283.

rences are frequent at these periods. In cases of chronic gout the patients are usually much better during the summer (especially if hot and dry) than in the other seasons of the year.

There can be no doubt that *errors in diet* are the most potent cause both of functional derangement of the liver and also of gout, and that when, as too often happens, deficient exercise is superadded, the development of the gouty diathesis is in many cases only a question of time. Many people habitually take much more food, especially of the albuminous kind, than the system requires ; a portion at least of this excess, after conversion into peptone, is absorbed into the blood, and conveyed to the liver. Moreover, the presence in the stomach of an excessive quantity of peptone interferes with the dissolving action of the gastric juice, and imperfectly digested substances are liable to be absorbed. Congestion and enlargement of the liver, a state of general plethora, excessive formation of uric and other acids, and derangement of the eliminative functions of the kidneys are the ordinary consequences. Even after the moderate use of meat, there is increased excretion of uric acid, as compared with the results of fasting, and when

albuminates are taken in excess the increase is very considerable. Many persons who eat immoderately also take an insufficient amount of exercise, and when these two conditions are associated it is difficult to define the share which each takes in the production of disease. The disproportion, however, between the absorbed albuminates and the absorbed oxygen must result in imperfect oxidation, and its consequences; the most important of which are retention in the system of refuse materials, and irritation of the eliminating organs (*e.g.*, the kidneys and skin) by the passage through them of excreta insufficiently prepared for removal from the system. The digestibility of the food is also a very important point in reference to the causation of gout. Rich, highly-seasoned dishes, by causing indigestion, aggravate the effects of an excessive quantity of food. It must be admitted that large meat eaters do not invariably suffer from gout, and that they sometimes exhibit no signs of obesity. If a *large* amount of exercise be regularly taken, the excess of albuminates may perhaps be consumed without causing any functional derangements; and in some cases, where much food is taken, a large proportion is insufficiently masticated

and is not absorbed, but discharged with the fæces. Exercise will not necessarily counteract the effects of excessive indulgence in the pleasures of the table. As Sir Thomas Watson points out "Gout used to be exceedingly common in the old-fashioned fox-hunter, who 'rode hard' while he also 'lived hard.'" Albuminuria is a not unfrequent result of indulgence in excess of albuminous food; some portion of the excess, it may be presumed, being thus got rid of.

In connection with this subject the following observations of Dr. Carpenter* appear to be very apposite:—"It is worthy of remark that in the times when even the wealthy lived during four or five months of the year almost exclusively upon meat, bread, and flour puddings, and when, therefore, the diet was far too highly azotized, as well as deficient in fresh vegetables, arthritic, calculous, and scorbutic disorders were much more common than at present. The introduction and universal employment of the potato has unquestionably done much to correct these two tendencies: on the one hand, by diluting the azotized constituents of the food, so that with the

* "Principles of Human Physiology," ninth edition, p. 100.

same bulk a much smaller proportion of these is now introduced; and on the other by supplying to the blood some element which is essential to the maintenance of its healthy condition. But with the diminution of the Arthritic diathesis, which the experience of our older practitioners and the medical writings of the last century indicate as having taken place during that period, there has been an increase n the Rheumatic; a change which seems to have a close relation to this alteration in diet."

Opinions differ considerably as to the influence of the other alimentary substances in the causation of gout. Sir A. Garrod's* views upon several of these points are somewhat at variance with those usually held, for he appears to think that *sugar*, for example, inasmuch as it has no influence upon the production of uric acid, may be allowed in cases of gout. It is, on the other hand, *generally* believed that sugar, unless in the most moderate quantities, is almost poisonous to a gouty subject. This point has an important bearing upon the question of diet, and it is therefore neces-

* Lumleian Lectures, *Medical Times and Gazette*, 1883, vol. i., p. 553. Free use has been made of these lectures in the preparation of several of the following paragraphs.

sary to discuss it somewhat minutely. There are three kinds of sugar met with in food—cane-sugar, grape-sugar or glucose, and milk-sugar or lactose. Cane-sugar when taken into the stomach is converted into glucose, and this substance is very liable to undergo the alcoholic fermentation. Milk-sugar, on the other hand, is much less liable to this change, but in the presence of almost any nitrogenous body it is very prone to undergo conversion into lactic and butyric acids. The fermentation of milk-sugar is especially apt to occur in the presence of cheese. It is a well-known fact that when starchy articles of food are taken, the starch is converted into glucose by the action of the salivary, pancreatic, and intestinal secretions, and it is therefore obvious that amylaceous materials may supply the system with large quantities of sugar. Sir A. Garrod doubts whether sugar causes what is popularly termed acidity, and in advising or prohibiting the use of sugar it is important to discover whether there is any real foundation for the *popular* belief on this point. By some persons lumps of sugar are taken to prevent heartburn, and, as Sir A. Garrod points out, it is scarcely credible that a little cane-sugar would seriously add to the glucose

which is daily produced in the alimentary canal of an individual living on an ordinary mixed diet. It is tolerably certain that the use of a large quantity of sugar does not increase the amount of uric acid in the urine; but, on the other hand, the influence of saccharine and starchy materials upon the production of fat is well known. These materials are easily oxidised, and by virtue of this property they delay or prevent the normal disintegration of the albuminous constituents of the body. It has also been supposed that the taking of sugar causes the uric acid to assume a less soluble form, but it has not been proved that the acidity of the urine is increased after the use of sugar in any form. It would therefore seem that the evil effects of saccharine substances in gouty subjects are due in great measure to the fact that they retard metamorphosis. If sugar or starch be taken in excess, the urine often becomes saccharine.

The remark just made with regard to the influence of saccharine and amylaceous food in retarding metamorphosis, applies also to the fatty and oleaginous group. The free use of these substances promotes obesity and prevents waste of tissue, but does not appear to influence in any degree the elimination of

any constituent of the urine. Fatty substances are, however, very prone to undergo butyric fermentation.

The "acidity" so often complained of by gouty subjects is the result partly of the fermentation of food and partly of the increased secretion of gastric juice. The most obvious symptoms of this condition are a sour taste in the mouth, acid eructations, acid reaction of the saliva, and sour odour of the breath. There is no doubt that in many cases the nature of the food greatly influences the degree and character of the acidity. A diet consisting chiefly of vegetables is, as a general rule, much more productive of this symptom than one in which animal food largely preponderates. A reason for this difference could be easily found if it be assumed that by a process of fermentation, glucose becomes converted into lactic acid, the presence of which is the cause of the acidity. Glucose, however, is generally much more prone to undergo the alcoholic than the lactic fermentation, although it is theoretically easy to show that one part of glucose is equivalent to two parts of lactic acid. This latter, again, is readily converted into butyric acid, which, in cases of heartburn, is often abundantly present in the stomach. It is, however, very probable

that sugars and starches often undergo lactic fermentation in the stomach, the gastric mucus, especially when produced in abnormal quantity, acting as a ferment. The albuminous substances are capable of undergoing butyric fermentation, either with or without previous lactic fermentation, and other acids, such as the acetic, succinic, etc., are often developed at the same time.

The next point for consideration, namely, the influence of *alcoholic liquors* in the production of gout, is one of great importance. These beverages are usually classified under the heads of spirits, wines and malt liquors. It would seem that alcohol, taken in the form of brandy, whisky, gin, etc., cannot be regarded as a cause of gout, inasmuch as in spirit-drinking countries the disease is almost unknown. The immoderate use, however, of distilled spirits is a common cause of disease of the liver and kidneys; and where the gouty predisposition exists, any habit which tends to damage the kidneys must augment the constitutional disorder. The effect of wine is more easily demonstrated, but it varies very much in degree. Full-bodied wines containing much unfermented matter are potent for evil in persons of a

gouty habit, and will doubtless produce the disease in a large proportion of cases in which there is no gouty taint. The light, well-fermented wines, on the other hand, are not liable to produce gout. The evidence that the disease is often traceable to the use of malt liquor is extremely strong. The occurrence of gout among the poorer classes is, in the absence of hereditary predisposition, almost always due to excessive consumption of beer. Sir Thomas Watson alludes to the prevalence of gout among the servants of wealthy families.

The case with regard to alcohol as a cause of gout may therefore be summed up by saying that distilled spirits and the lighter wines appear to be innocuous in this direction, while malt liquors of all kinds, and the stronger and imperfectly fermented wines (comprising port, sherry, madeira, and champagne), play a very conspicuous part in the causation of gout. We have no definite knowledge as to the mischievous principle; it may be, as Sir A. Garrod believes, a something which is the result of imperfect fermentation, for it is certain that those beverages in which fermentation has been prematurely checked are much more liable to produce gout and gravel

than those in which the process has been allowed to attain completion. The natural acidity of wines plays a very small part in the production of the gouty diathesis; but that acid wines often excite a paroxysm in gouty subjects is a matter of common observation.

The influence of *lead* as a predisposing cause of gout is a subject of considerable interest, for there can be no doubt as to the frequency of the coincidence of lead-poisoning and gouty manifestations. In many of the recorded cases it is probable that other factors assisted in the production of the disease; but cases of gout for which no other causes can be discovered are occasionally noticed. It is somewhat curious that in France, where lead-colic is common, gout is a very rare disease, and this fact lends weight to the supposition that the effect of the lead is often much heightened by indulgence in alcoholic liquors. The experience of French physicians is supported by that of Dr. Oliver,* of Newcastle-on-Tyne, who states that native lead-workers in the North of England seldom become gouty, even when the kidneys are affected. On the

* Goulstonian Lectures on Lead Poisoning in its Acute and Chronic Manifestations. *Brit. Med. Journal*, 1891, Vol. I., p. 619.

other hand, workmen coming from the South are apt to develop gout in the North of England. In lead-poisoning the blood and tissues are rich in uric acid, while the urine is deficient in this substance. It is doubtful whether there is increased production of uric acid or defective elimination, but the latter condition would appear to prevail. Dr. Haig states that the primary action of lead is to diminish the solubility of uric acid, to bring excretion below formation and to retain it in the body. "Then when from dyspepsia or intestinal pain (due to urate of lead in the intestinal walls), the urea and acidity fall, the retained uric acid is washed out into the blood-stream, and uric-acidæmia, with slow, high tension pulse, accompanies and follows the lead-colic." It is a remarkable fact that contracted kidney occurs in not a few cases of chronic lead-poisoning, certainly in too large a proportion to be regarded as a mere coincidence. There is another curious relation between lead-poisoning and gout, for it is highly probable that persons of a gouty habit are more liable than others to suffer from the effects of the mineral. Even in medicinal doses salts of lead are wont to cause very marked symptoms in gouty subjects.

There is one other predisposing cause of gout which deserves a passing notice, and especially because it is connected with one of the current theories of the nature of the disease. Sydenham found that, in his own case, an attack of gout could be excited by severe study, and in some gouty persons anxiety of mind, grief, or annoyance produce the same effect. It is, however, scarcely credible that mental influences alone should suffice for the *development* of the gouty diathesis. Severe study is often associated with sedentary habits and errors in diet, which, as I have just attempted to show, are potent causes of gout. With regard also to other depressing influences, such as exposure to cold, sexual excesses, and the like, these no doubt often excite a paroxysm, and may even aid in the development of the diathesis; but in this latter respect they are not to be compared with the causes already discussed.

A few words as to the *exciting* causes of the paroxysms will conclude this portion of the subject. Experience teaches us that wherever the predisposition exists, almost anything, whether of an exciting or debilitating character, that disturbs the health, is capable of inducing an attack. The most common

*exciting* causes are therefore excesses in eating and drinking, indigestible food, articles of diet which disagree with the patient's idiosyncrasy, exposure to cold, excessive mental exertion, violent bodily exercise followed by fatigue, mental excitement, venereal excesses, debilitating diseases, loss of blood, etc. The action of one at least of these causes is very generally recognised. In some gouty subjects pains in the joints, or even an acute attack, are the ordinary results of taking even two or three glasses of port or champagne. In addition, however, to causes of a more or less general nature, it not unfrequently happens that local injuries, whether of a severe or slight character, are sufficient to induce an attack. Thus operations and fractures of various kinds, bruises, sprains, and even the wearing of a tight boot, may act as exciting causes. In gouty subjects very slight local injuries often give rise to unusually persistent and severe pain which is not relieved by ordinary remedies, but subsides under appropriate treatment. Acute gout, moreover, sometimes attacks a joint which has suffered in some previous injury. In another class of patients, such a change of habits as is involved in confinement to bed for a few days is

sufficient to provoke an attack of gout. My friend Dr. Gordon informs me that, when in medical charge of a jail, he not unfrequently witnessed the development of acute articular gout in prisoners whose course of hard labour had been suddenly interrupted by an attack of illness necessitating confinement to bed.

# CHAPTER V.

## IRREGULAR MANIFESTATIONS OF GOUT—VISCERAL AND CUTANEOUS AFFECTIONS.

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Irregular Gout—Suppressed Gout—Peculiarities of the Irregular Manifestations—Detection of Uric Acid in the Blood in these Cases—Visceral Gout—Functional and Organic Affections—Gouty Diseases of the Throat and Stomach—Sir M. Mackenzie's Cases of Gouty Throat—Misplaced Gout of the Stomach—Retrocedent Gout of the Stomach—Two Forms: Spasmodic and Inflammatory—Dr. Buzzard on Probability of mistaking Gastric Crises of Tabes for Gout of the Stomach—Question of Metastasis—Experimental Production of the Phenomena of the Uric Acid Diathesis—Gouty Disorders of the Intestines: Dr. Haig's Views—Gouty Affections of the Heart—Retrocedent Gout—Condition of Heart in Fatal Cases—Fatty Degeneration—The Gouty Heart—Gouty Phlebitis—Gouty Affections of the Lungs: Asthma and Bronchitis—Dr. Stokes' Views—Dr. Greenhow's Cases—Gouty Affections of the Nervous System—Gout Retrocedent to the Brain—Epilepsy, Insanity, Neuralgia, Headache, etc., in Gouty Subjects—Gouty Neuritis and Paralysis—Gouty Affections of the Eye—Mr. Hutchinson and Mr. Brudenell Carter's Views—Gouty Affections of the Ear—Disturbances of the Sensorial Functions—Gout and Eczema—Dr. Piffard on the Connection between certain Skin Diseases and the Rheumatic Diathesis.

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There is no difficulty in recognising the paroxysms of *regular* gout. The suddenness of the attack in

the majority of cases, the seat and character of the pain, and the condition of the joint are sufficient to determine the diagnosis. There are, however, many other phenomena connected with the disease, but of a far less definite character, though due to the same cause, viz., the presence in the blood of excess of sodium urate. I propose to discuss, without unnecessary detail, the most important of these manifestations, to which the term *irregular* may be conveniently applied.

In former times a vast number of symptoms were referred to gout. At the present day a very different tendency prevails, and we hear more about "neurotic conditions," "trophic nerves and lesions," and the like. Some modern authorities indeed assert that the term "gout" is very often a refuge for ignorance; implying that when no other cause can be discovered the phrase "suppressed gout" is clutched at in desperation, in order to evade a serious difficulty. Crabbe had doubtless observed several instances of this kind when he wrote in *The Library*,—

> "One to the gout contracts all human pain;
> He views it raging in the frantic brain;
> Finds it in fevers all his efforts mar,
> And sees it lurking in the cold catarrh."

There is probably some amount of truth in these insinuations, but at the same time it is not unreasonable to assume that the same cause or causes which give rise to *regular—i.e., articular*—gout are capable of producing certain effects or symptoms in parts of the body other than the articular structures. It must, I think, be admitted that a high degree of probability is attached to this supposition, and that it is perfectly justifiable to speak of *irregular* manifestations of gout. It is a matter of common experience that certain symptoms, such as those already mentioned as connected with the uric acid diathesis, often precede the acute attack, disappear on its occurrence, and again make themselves felt some time after the articular symptoms have subsided. The relief experienced after an acute attack is often very marked. We also find that many patients exhibit symptoms of the diathesis, but never suffer from decidedly acute attacks, and there is no escape from the conclusion that both classes of manifestations are due to the same cause. It is not of course contended that all symptoms which may occur, even in decidedly gouty patients, are necessarily due to the diathesis ; but it is extremely probable that a gouty taint modifies the

course and symptoms of any disorder which affects its subject.

In cases at all urgent, before arriving at a conclusion with regard to symptoms suspected to be due to gout, it is well to adopt Sir A. Garrod's suggestion and examine the blood-serum obtained from a small blister in the manner already described. If crystals of uric acid are discovered it is highly probable that the suspicion is correct. As a matter of course, the age, sex, previous history, habits of life, and other circumstances must be duly considered. The previous occurrence of an acute attack of gout is clearly suggestive of the causation of anomalous symptoms, subject to the caution given in the preceding paragraph.

In my first chapter I cited Cullen's classification of the various forms of gout. The terms *atonic* and *wandering* are at the present day almost obsolete ; we now speak of *visceral* gout, which includes *misplaced*, *retrocedent*, and *atonic* forms. This collective term is applied to designate all symptoms having their seat in internal organs, and presumably due to the influence of the gouty diathesis. The visceral affections are again further subdivided into functional and organic.

An important addition must, however, be made to this category, for, as I shall presently endeavour to show, certain skin-affections are closely connected with the gouty diathesis, and may, indeed, be reckoned among its most important manifestations. In the present chapter I propose to deal with these affections and with gouty disorders of some of the thoracic and abdominal viscera, leaving, however, gouty diseases of the liver and kidneys for a subsequent chapter.

Symptoms of *visceral* gout may precede the articular inflammation, may co-exist with it, or may suddenly appear on its subsidence. Many of the symptoms of the uric acid diathesis (see page 55) may be regarded as those of visceral gout in an incipient stage. Gouty disorders of the *digestive organs* are common precursors of an attack of gout, and in my experience often occur alone, that is, without any decided articular affection. The throat is not unfrequently affected in gouty patients, the symptoms of angina sometimes alternating with attacks of inflammation of the joints. In the case of a gentleman, aged sixty-five, under my care some years ago, the almost sudden subsidence of an acute attack of gout in the toe was followed by acute inflammation of the

pharynx, lasting for several days, and attended by much constitutional disturbance. He has subsequently had several attacks of acute gout, running the ordinary course, but not accompanied by any throat-affection. The late Sir Morell Mackenzie* recorded several well-marked instances. "In one case, a gentleman who frequently suffered from attacks of angina became subject to gout, and was never again attacked with inflammation of the throat. In another case, the patient was suffering from acute pharyngitis, when the symptoms suddenly disappeared, and an acute attack of gout developed in the great toe of the right foot; after three days the gouty inflammation of the toe disappeared and acute hyperæmia of the pharynx supervened."

Gouty subjects still more frequently suffer from chronic catarrh of the fauces and naso-pharynx. The mucous secretion is apt to accumulate during the night, and is got rid of with some difficulty. The catarrh may extend to the larynx and trachea, producing hoarseness and a troublesome cough. Sir Dyce Duckworth says "The gouty throat is like no

* "Diseases of the Throat and Nose," vol. i., p. 48.

other. The pillars of the fauces, especially the posterior pair, the velum and the uvula are very red and glazed. . . . The uvula is greatly enlarged and elongated."

Dyspepsia in various forms—acidity, flatulency, irregular action of the bowels—and hæmorrhoids, are common symptoms in gouty subjects and in persons in whom the hereditary tendency is well marked. Their connection with gout is rendered probable, first by their subsidence on the occurrence of an acute attack, and, secondly, by the manner in which they are influenced by regimen and medicines suitable to the gouty diathesis. Charcot records a case which illustrates the first of these characteristics. A patient, who had suffered from articular gout, became a martyr to dyspepsia, and, after vainly invoking the aid of regular science, had recourse to homœopathy. The dyspepsia rapidly subsided, and the patient was congratulating himself on his change of advisers when a sharp attack of articular gout served to explain the supposed cure. A somewhat interesting case of a similar kind has recently been under my care. A gentleman, aged forty-six, has suffered at intervals during several years from attacks of violent palpitation of the

heart, coming on suddenly at night, and accompanied by "rushing of blood to the head" and a feeling of impending dissolution. The attacks are followed and relieved by eructation of flatus from the stomach; for some days afterwards vertigo, numbness of the hands and feet, and sensations of "pins and needles" are more or less troublesome. The nocturnal suffering can be almost invariably traced to indulgence in the pleasures of the table, including champagne. The urine is always highly acid, and a copious deposit of uric acid takes place after these attacks. All the symptoms subside under appropriate treatment; and on two occasions a similar result was witnessed immediately after the outbreak of acute gout in the great toe.

When acute gout attacks the stomach, instead of the structures of a joint, it sometimes proves rapidly fatal. As a general rule, the patient has suffered from ordinary gout for some years, and from more or less acute attacks of gastric derangement. Without further warning a burning, gnawing pain is felt in the pit of the stomach, and rapidly increases in severity. The pulse becomes very small; the skin is cold and covered with perspiration, and there are signs of

approaching collapse. Attempts at vomiting only aggravate the condition, which resembles that produced by irritant poisons. In one case of this kind, terminating fatally, the late Dr. Sutton thought that he "could see lithate of soda deposit distinctly in the wall of the stomach" on post-mortem examination. When called to a case presenting the symptoms above described, it is well to remember the hint given by Sir T. Watson, and to inquire whether any indigestible food has been lately taken, "for *gout* (so-called) *in the stomach* has sometimes turned out, under the test of an emetic, to have been nothing more than *pork in the stomach*." Sir T. Watson, however, states positively that gout may attack the stomach, and that the symptoms may point either to spasm or to inflammation.

More important, in some respects, than this *misplaced gout* of the stomach are those attacks to which the term *retrocedent* is commonly applied. It is impossible to suggest any really satisfactory explanation of the so-called "metastasis," but cases undoubtedly occur in which violent gastralgia, vomiting, prostration, or even death, abruptly supervene upon the subsidence of the symptoms of articular gout. In

some cases the metastasis is spontaneous; in others, it appears to result from attempts to relieve the articular inflammation by local remedies, or from exposure to cold or other injurious influences, during the course of an acute attack. The effects of exposure to a chill are well illustrated by the following cases. A lady, aged fifty-four, who, since the menopause, had had several acute attacks of gout, was exposed to cold and damp when threatened with a renewal of the joint-symptoms. These subsided, but vomiting and other symptoms of acute gastric irritation set in, and were not relieved until the gout was developed in the toe, some thirty hours afterwards. In another case which came under my notice, a gentleman, aged forty-four, suffering from acute gout, applied cold wet cloths to the joint. Symptoms of acute gastritis rapidly supervened, and were followed by syncope and death in seventeen hours.

Cold applications to the affected joint or placing the foot in cold water will often relieve the local symptoms, but at the cost of serious gastric or cardiac disorder, and a full dose of colchicum has also been known to produce similar results.

Gout affecting the *stomach* presents at least two

forms, which may be termed respectively *spasmodic* and *inflammatory*. In the former, which may be regarded as a functional affection, there is violent spasm or cramp in the epigastric region, accompanied by a feeling of intense oppression and distension, nausea, vomiting, difficulty of breathing, and palpitation of the heart. There is also great prostration; the skin is cold and clammy, and the pulse is feeble, frequent, and irregular. There is, however, no severe burning pain, and the cramp is relieved by pressure and stimulants. In the inflammatory form the symptoms are those of acute gastritis. There is severe epigastric pain of a burning character and much aggravated by pressure, nausea and vomiting, and more or less febrile disturbance, and the ejected matters sometimes contain blood. The pulse is at first full and strong, but soon becomes feeble and irregular. Stupor is apt to supervene, and the patient becomes almost insensible; but slight pressure upon the epigastrium calls forth evidences of severe pain. These symptoms, like those of the spasmodic form, may either suddenly subside, or terminate in death. Recurrence of the articular inflammation is sometimes simultaneous with the abatement of the gastric symptoms.

Certain phenomena much resembling those of acute

gouty disorder of the stomach are not unfrequent in cases of locomotor ataxy. Dr. Buzzard,* indeed, in his excellent Lectures, suggests "that many cases of so-called 'gout in the stomach' would be found, if examined by the light of our present knowledge, to be examples of tabes dorsalis, with gastric crises. The lightning pains, which would probably be associated with the sickness and epigastric pain, would be likely in the minds of many to make the diagnosis of 'gout in the stomach' complete." Severe gastric attacks, it must be remembered, are sometimes witnessed in persons who, although examples of tabes dorsalis, "show at the time no sign of inco-ordination of movement." Their persistence and frequent recurrence would suffice to exclude gout as a cause, and if accompanied by absence of patellar tendon-reflex and by other indications of tabes, there ought to be no difficulty in the diagnosis.

This subject of metastasis, or transfer of diseased action, is involved in much obscurity, and not a few authorities assert that symptoms such as those above described are capable of a very different explanation.

* "Clinical Lectures on Diseases of the Nervous System," p. 272.

The old notion of metastasis was to this effect—a local irritation, due to some permanent general diathesis, becomes cured or subsides, but throws itself on some other part in which it excites symptoms of disordered action or of inflammation. That this is the *apparent* course of events there can be no doubt, but as to the manner in which the irritation is propagated we can only speculate. It is very probable that in many instances of supposed metastasis, the occurrence, *e.g.*, of gout in the stomach, was a mere coincidence, and that the symptoms were only those of severe indigestion, to which gouty subjects are especially liable. Chronic gastritis is a common condition in these patients, and the symptoms of gastric disorder are easily aggravated, and become prominent under the operation of comparatively slight causes. Admitting, however, that in some cases the phenomena of retrocedent gout, or gout of an internal organ, are due to a previously existing affection, it is going too far to assert that all instances of retrocedence can be thus explained. Charcot points out that phenomena analogous to those of the uric acid diathesis can be experimentally produced in animals, and that in such cases the gastric juice and the follicles of the stomach are

loaded with sodium urate. He thinks that, without assuming the existence of an identical condition to account for the symptoms of stomachic gout, superficial lesions might be easily caused in the digestive system under the influence of retrocession. However this may be, "it is probable that in the long run permanent lesions are produced in the cases in which these manifestations occur, which seem so purely functional."*

*Gouty disorders of the intestines* have seldom been recognised, and some authorities would probably deny that the intestinal tube is liable to be thus affected. My own experience, however, has led me to form a different conclusion; I have met with several cases of colic and enteralgia in gouty subjects, and have found that speedy relief was obtained from remedies which promote the elimination of uric acid. In one case, that of a gentleman aged 68, the patient had for some twenty years been subject to an annual attack of gout. Not long ago, at the time when the usual articular symptoms might have been expected, an attack of acute pain in the cœcal region suddenly

* Charcot, "Lectures on Senile Diseases," New Syd. Soc. Trans., p. 85.

came on, and was accompanied by vomiting and marked elevation of temperature. Before the attack the urine generally contained excess of urates and much free uric acid; but as the acute symptoms passed off, the uric acid disappeared. Relief was speedily obtained from the administration of antipyrin, sodium salicylate, and sodium iodide in combination. With regard to this case, it may be added that a recurrence of the intestinal symptoms took place last year, with a fatal result after three days. The patient was not under my care; the attack came on when he was at some distance from London, and fuller details could not be obtained.

In another case, that of a lady aged 54, whom I had treated 18 months previously for an attack of gout in the toe, the symptoms were manifested in the abdomen. There was severe pain, constant sensation of nausea, complete anorexia and occasional vomiting. The stomach was not appreciably dilated, but there was a great deal of flatus at times and distressing borborygmi, accompanied by marked febrile movement. There were indications of slight congestion of the liver, and pills containing colocynth, blue pill and henbane were accordingly administered.

Full doses of alkalies were then given, and oxalate of cerium to control the vomiting. The strength was sustained by zyminized suppositories, administered every four hours. After the acute stage had subsided, salol was given. The effect of the treatment upon the condition of the urine was very marked. The secretion had been scanty and high-coloured, free from albumen, but with a copious deposit of lithates. It subsequently became more abundant, and free from any excess of lithates. The acidity also greatly diminished; expressed in terms of oxalic acid it was only 3·8 parts per 1,000, and later on it fell to 2·8 parts per 1,000. The pain and vomiting entirely ceased, and the patient was soon able to take a fair amount of nourishment. Complete recovery took place.

My opinions founded on these cases and others of a like character have been greatly strengthened by a perusal of Dr. Haig's paper* on "Gout of the Intestines." His description of his own symptoms and of the manner in which they were relieved by salicylate of sodium is quite applicable to several of

* *The Practitioner*, Jan., 1893. p. 17.

my patients. Dr. Haig's view of the pathology of such cases is extremely interesting. He believes that certain factors, notably various drugs such as mercury and lead, and likewise acids and exposure to cold, diminish the excretion of uric acid in the urine. They "clear it out of the blood and drive it into the liver, spleen and fibrous tissues, especially into any fibrous tissues that have had their alkalinity diminished by previous irritation or inflammation. They produce this effect on uric acid, either, as in the case of metals, by forming insoluble compounds with it, or by diminishing the solvent powers of the blood through a lowering of its alkalinity; and under slightly different conditions any one of them may produce gout of a joint instead of gout of the intestines."

The walls of the intestines contain a large amount of fibrous tissue, and if the alkalinity of this tissue be diminished by injury or irritation, a deposit of urates in the walls of the bowels is by no means unlikely to occur, just as in the case of articular structures. Sir Dyce Duckworth* cites Professor Hayem's account of a case of enteritis in which the villi were found to

* "A Treatise on Gout," p. 89.

be strewn with small uratic incrustations. Sir A. Garrod also mentions the case of a very gouty man, in whom, after exposure to cold, gout retroceded to the intestines, producing intense inflammation of the last eighteen inches of the ileum, as found on post-mortem examination. Dr. Haig explains the special liability of the lower part of the ileum and the cœcum to gouty disorder by suggesting that alkalinity is diminished in the small intestine before the ileo-cœcal valve is reached. He thinks that irritation in the vermiform appendix may precipitate a local attack of gout which, as in other parts—the joints and valves of the heart, for instance—recurs and recurs till a more or less extensive lesion (perityphlitis) has been produced. In connection with this subject it may be mentioned that Dr. Bence Jones has recorded a case of lithæmia with violent cramps in the rectum as a prominent symptom; these came on six or eight hours after food and lasted from half-an-hour to an hour. They entirely ceased under careful diet and the use of alkalies.

Of the organs of the *thorax,* the *heart* often becomes affected in cases of gout, and the disorder may be either functional or organic. Functional dis-

order is evidenced by palpitation, precordial pain and a feeling of oppression, more or less dyspnœa, anxiety, feebleness and irregularity of pulse. These symptoms are in many cases merely those of dyspepsia, or of an overloaded or distended stomach, and are in no way characteristic of gout. They, however, frequently accompany the development of the uric acid diathesis, and, in some cases at least, are probably due, not directly to indigestion, but to the action upon the heart of the sodium urate contained in the blood. When an acute attack occurs in a joint, these cardiac symptoms not unfrequently subside, but only to reappear later on, thus alternating with the joint-affection.

In considering retrocedent gout affecting the heart, the difficult question of metastasis again comes before us. Cases, however, now and then occur in which the morbid action *appears* to be transferred from the joint to the heart. I have already referred to a case in which gastric symptoms were followed by fatal syncope. In one recorded case a patient suffering from severe articular gout applied snow to the affected part. The pain was relieved, but marked symptoms of syncope came on, and the patient was for some time in a

very critical state. In another case, under the care of Sir A. Garrod,* the cardiac symptoms followed exposure to cold east winds at a time when the patient was recovering from an acute attack. There was violent pain across the chest and down both arms, with faintness and a sense of alarm. The symptoms, after partial subsidence, recurred several times, and a few days after their final disappearance the foot again became affected. "There was no evidence of structural disease of the heart, and no appreciable febrile disturbance."

Whatever may be the true explanation of these and similar cases, the condition of the heart, when death occurs from retrocedence of gout to that organ, is always sufficient to account for the symptoms and the result. Fatty degeneration or fatty deposit, with or without valvular disease, and dilatation, are invariably present. An increased amount of fat on the surface of the heart, accompanied by atrophy of the muscular tissue, is common in gouty persons presenting external signs of obesity. In all these conditions, shortness of breath, or a sense of choking or suffoca-

* "Gout and Rheumatic Gout," third edition, p. 440.

tion, a tendency to faintness, especially on exertion; a feeble cardiac impulse, and a small, slow, and irregular pulse, are usually more or less prominent symptoms, but it is well known that these are sometimes absent in cases in which extensive degeneration is discovered after death. In other cases there are symptoms of angina pectoris, which in gouty subjects is almost always accompanied by organic changes. This affection of the heart is not unfrequently connected with gout. The two complaints sometimes alternate with each other in the same patient; and in such cases, as pointed out by Dr. Latham,* in all probability the angina has been a purely functional affection, the heart suffering pain and spasm, though perfectly sound of structure. "That such an angina should germinate from the same root as gout is not unlikely. They might both spring from some inveterate fault of the assimilating processes."

When retrocedent gout attacks the heart, already in a state of fatty degeneration, rupture of the organ is the most frequent cause of a suddenly fatal issue. Death may also occur from syncope, without rupture.

* "Collected Works," New Syd. Soc. Ed., vol. i., p. 480.

If it be admitted that retrocedent gout may give rise to symptoms of cardiac disturbance in the absence of structural diseases of the organ, it is only reasonable to expect that the same cause would produce much more serious consequences when organic lesions are present. It is not necessary to assume that gout sets up any kind of inflammation of the heart, but those factors which are most potent in the development of gout tend also to produce fatty changes in the organ. Gout, moreover, as I shall presently attempt to show, plays an important part in the causation of Bright's disease, and is thus again connected with cardiac disorders. Gout also appears to be one predisposing cause of atheroma, which, when it affects the coronary arteries, often gives rise to fatty degeneration.

The condition termed the "gouty heart" has attracted much attention since the middle of the present century. Dr. Bright, indeed, in 1837, when describing the disease which bears his name, remarked that "the deviations from health in the heart are well worthy of observation; they have been so frequent as to show a most important and intimate connection with the disease of which we are treating." He

offered two explanations of the ventricular hypertrophy associated with renal disease: (1) that the altered composition of the blood afforded an irregular and unwonted stimulus to the heart itself; or (2) it so affected the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system. The second hypothesis is now generally accepted, but pathologists are by no means agreed as to the cause of the obstruction. Sir G. Johnson pointed out that the arterioles of the kidneys and other parts displayed a thickening of the muscular coat, by which the blood-flow was obstructed. On the other hand, the late Sir W. Gull and Dr. Sutton described a condition of arterio-capillary fibrosis associated with "disease in the connective substance of the wall of the left ventricle, a similar process of degeneration to that seen in the contracted kidney itself and in the blood-vessels." According to these observers, the kidneys may be little if at all affected, whilst the morbid change is far advanced in other organs. This is not the place to enter at any length upon the pathological questions at issue; it is only necessary to describe in a few words the process of development of the cardiac lesions.

A condition of lithæmia is the starting-point of the pathological changes. Waste products of various kinds are in excess in the blood, and evidence of high arterial tension is obtainable both by ordinary examination of the pulse and by the sphygmograph. The pulse at the wrist is full, hard, and only slightly compressible. Contraction of the hypertrophied arterioles causes persistently high arterial tension and an obstacle to the outflow of blood from the left ventricle, which is imperfectly emptied and subjected to increased blood-pressure on diastole. Hypertrophy of the ventricle is sooner or later developed. This first stage of lithiasis is most often seen in men of middle age, who appear to be well nourished, and whose brains are active and constantly at work.

On examining the heart in these cases, the first sound is heard to be prolonged and muffled, *i.e.*, less clear than usual, and the aortic second sound is accentuated and sometimes of a ringing or metallic character. The triangular space of complete cardiac dulness is enlarged, and there may be displacement of the apex outwards and a heaving impulse. The sphygmograph shows exaggeration of the systolic upstroke and retardation of the diastolic downstroke; in-

creased pressure is needed to bring out the characters of the pulse. When marked atheroma is present, the tracing will often be that described as "square-headed." Dilatation is apt to accompany the hypertrophy, and palpitation is then a frequent symptom. Disease of the aortic valves may supervene; mitral disease is less frequent in these cases. The lesions of the kidney associated with those of the heart will be described in a subsequent chapter.

In connection with the circulatory organs, gouty *phlebitis* requires to be briefly noticed. Inflammation of the veins, especially in the lower limbs, is somewhat liable to occur in gouty subjects, and either with or without acute attacks in the joints. A slight blow or prolonged friction may act as the exciting cause. The superficial veins are those which are most frequently affected; the inflammation begins very suddenly, and causes more or less severe pain and tenderness. In other cases, the process is of an insidious character, and is attended with comparatively slight uneasiness, though resulting in the formation of an extensive and firm thrombus. The inflammation may spread continuously, and may appear simultaneously in several veins. It sometimes disappears

and affects other veins at a distance. Sometimes the deep veins are affected, in which case the limb becomes œdematous and distended. Recovery is often tedious, and the inflammation is apt to recur. An eruption of eczema often increases the patient's troubles. Symptoms of embolism have been known to supervene, with death from obstruction of the pulmonary artery as a result. A case of apparently gouty embolism of a pulmonary vein, attended by very alarming symptoms, has lately been recorded by Dr. J. Gordon Black, of Harrogate. The patient, aged seventy, who had never suffered from acute articular gout, was suddenly attacked with severe pain and swelling in the right knee-joint. Two days afterwards œdema of the left lung became suddenly developed, and the patient's life was in great jeopardy. The acute symptoms subsided under the free use of ether hypodermically. Sir James Paget* says that gouty phlebitis is often hereditary, and that common phlebitis is often traceable to the gouty diathesis. He thinks that gout should always be suspected whenever phlebitis occurs without evident external cause in an

* "Clinical Lectures and Essays," p. 300. See also Sir P. Hewett's Address in Clin. Soc. Trans., 1873.

elderly person. The late Dr. Owen Rees thought that gout was essentially a *capillary phlebitis,* and that this theory best explains the phenomena of the acute paroxysm, the venous inflammation being caused by the circulation of a blood-poison.

If we turn to diseases of the respiratory organs, we find that *asthma* and *bronchitis* are often connected with the gouty diathesis. In some cases of gouty asthma, there is no organic affection of the respiratory apparatus, while in others bronchitis or emphysema co-exists. The connection between the asthma and the diathesis is shown by the fact that the former either subsides or remits on the development of articular inflammation. Dr. Sutton gives the following illustration of the relation between the pulmonary symptoms and gout. "A man is seized during the night with an attack of asthma. He has some shortness of breath before going to bed, and it gets worse during the night; throughout the day the breathing is better, but we hear some bronchial râles; at night the asthmatic seizure returns. In a day or two an attack of gout comes on, and the lung-trouble disappears." Dr. A. T. H. Waters believes that lobar emphysema, resulting from malnutrition of the lung-tissue, is

sometimes associated with gout. It must not be forgotten that paroxysms of dyspnœa, closely resembling those of spasmodic asthma, are sometimes witnessed in cases of uræmia. In gouty patients with pulmonary symptoms, careful and frequent examination of the urine will aid in determining the nature of the complication.

There is still stronger evidence in support of the connection between gout and *bronchitis*. When we consider that many specific disorders are frequently accompanied by affections of the bronchial mucous membrane, it is not to be wondered at that a disease like gout should be liable to the same complication. I have witnessed the symptoms of acute articular gout disappear on the supervention of bronchitis. Crystals of uric acid have been detected in the sputa of a gouty patient,* and scarcely any stronger evidence could be offered of the occurrence of gouty bronchitis. There is every reason to believe that all the excretory organs take a share in the elimination of morbid materials, and that catarrh or inflammation is a frequent result. The proof that the bronchial symptoms are connected with

* See note by the late Dr. Hudson on page 81, New Syd. Soc. edition of Stokes, "On Diseases of the Chest."

gout is found in the fact that they often precede the attack and rapidly subside on its appearance, again to recur when the articular inflammation abates. In the case of a lady aged seventy, under my care a few years ago, attacks of acute gout had been very frequent for some time; bronchitis supervened and the gouty attacks diminished in number and severity. Dr. Todd* has recorded a case in which obstinate and severe bronchitis at once subsided on the appearance of gout in the foot; and Dr. Stokes alludes to cases in which bronchial symptoms having been removed by appropriate treatment, an attack of gout immediately appeared. He also refers to more complicated cases, such as the succession of epilepsy, gout, and fatal bronchitis; inflammation of the trachea, slight general arthritis, glandular enlargements, and gout. He doubts whether, in such cases, the anatomical characters of the lesion are different from those of ordinary bronchitis, and thinks that "its specific character will be found more in its mode of invasion and amenability to certain remedies than in its anatomical nature or seat." Valuable clinical

* "On Gout and Rheumatism," p. 38.

evidence with regard to the relation between a gouty constitution and bronchitis has been adduced by Dr. Headlam Greenhow.* He has given details of many interesting cases showing the frequency of gout among bronchitic patients; the frequent co-existence of gout and bronchitis in the same families; the alternation of the two affections in the same persons, and the frequent association of psoriasis, eczema, and gravel with bronchitis. Pleurisy and pneumonia are not unfrequent in gouty subjects, but it is doubtful how far the diathesis is concerned in their development. Emphysema is very often met with in cases of chronic gout.

Severe and well-marked gouty affections of the *nervous system* are of less frequent occurrence, but retrocedent gout sometimes attacks the brain and its membranes. The supervention of cerebral symptoms upon the sudden disappearance of gout from an extremity has been observed by Sir A. Garrod, and Niemeyer mentions two cases of gouty subjects presenting severe symptoms referable to circumscribed inflammation of the meninges. In one case the

* "On Bronchitis and the Morbid Conditions connected with it," second edition, p. 128, *et seq.*

symptoms disappeared simultaneously with a copious excretion of urates by the kidneys; and in the other, with an attack of gouty inflammation of the joints. Retrocedence of gouty inflammation, giving rise to apoplexy, has been known to follow the application of cold to a gouty joint, but as in the analogous case of the heart, it is probable that in the majority of instances, more or less severe organic disease had previously existed in the part. In another class of cases, symptoms resembling those of apoplexy pass off with the outbreak of typical arthritis. Less severe symptoms, such as slight stupor, temporary aphasia, defective mental alacrity, and impaired intelligence, are sometimes attributable to metastasis in gouty subjects. Epilepsy would appear in some cases to be connected with gout, the convulsions ceasing after the development of acute arthritic attacks.

Dr. Mortimer Granville* has pointed out that epilepsy, of gouty origin, is not unfrequent in women hereditarily disposed to gout. Sodium urate accumulates in the blood, either as a result of over-production or of non-elimination, and the accumulation finds its

* *British Medical Journal*, 1885, vol. ii., p. 701.

expression or outburst in the nerve storm. It is more than probable that a close association exists between gout and various forms of insanity. This connection was recognised by Sydenham, and is supported by the experience of many modern authorities. I am informed by my friend Dr. Newth, of Haywards Heath, formerly Medical Officer to the Sussex County Asylum, that a copious deposit of urates is a common symptom in insane patients, particularly during the subsidence of a violent attack. Symptoms of profound melancholia sometimes precede an attack of gout, and disappear as the joint-affection passes off. Moreover, in not a few cases, marked symptoms of mental disorder, without any external signs of gout, are relieved by treatment adapted to the uric acid diathesis. Dr. Maudsley says: "It can admit of no question that every degree of mental disorder, from the mildest feeling of melancholic depression to the extremest fury of delirium, may be due to the non-evacuation from the blood of the waste matters of the tissues." Hence he argues that there is a connection between gout and insanity, and he mentions the case of Lord Chatham as an example. Dr. Sutton cites the case of a patient with suicidal mania, confined in

an asylum ; the doctor finds it very difficult to prevent him from killing himself. "One morning when the doctor is going round the wards, the patient points to his toe and says, 'I am all right now, doctor;' he has had an attack of gout and his suicidal tendency is gone."

There is much reason for believing that many slight affections of the nervous system are referable to the presence of the gouty diathesis ; the term *nervous* gout has indeed been applied to the entire class of irregular gouty manifestations. Nervous affections of this character occur especially in women, and in individuals generally of a nervous temperament and descended from gouty ancestors. In some families the male members are the victims of acute gout, while the females suffer from neuralgia in various forms, headache, etc. As in other cases, the connection of any obscure symptoms with gout may be suspected when there is a history of the disease in the family and no other cause can be detected. When the symptoms alternate or co-exist with slight attacks of articular inflammation, there can be little doubt as to their origin. Cases are sometimes met with in which patients, once the subjects of frequent articular attacks, become

after a time exempt from them, but suffer instead from neuralgia, headache, sleeplessness, vertigo, dizziness, sensations of "pins and needles" in the hands and feet, and other symptoms of nervous disorder. Neuralgia is the most common symptom of nervous gout, and it most frequently appears in the form of hemicrania or megrim. In the case of a lady, aged sixty-seven, whom I saw with Sir William Jenner, gout and diabetes co-existed, and the patient suffered also from occasional attacks of megrim, with distressing sickness and intolerance of light. Dr. Liveing thinks there can be no question "as to the frequent connection of megrim, whether in its blind, sick, or simply hemicranial forms, with a gouty diathesis, and its occasional replacement by fits of regular gout. Megrim, however, is far from being the only neurosis which is thus associated with gout."* As stated in a previous chapter (see page 68), Dr. Liveing is inclined to think that gout in its various forms is the manifestation of a disorder which has its primary seat in the nervous system itself.

The close relation between some forms of headache

* "On Megrim, Sick Headache and some Allied Disorders," 1873, p. 404.

(notably megrim) and the excretion of uric acid has recently been demonstrated by Dr. Haig.* He found that he could cure headaches (to which he was remarkably subject) by the administration of an acid, which diminished the excretion of uric acid. Another effect was, however, produced, viz., pricking and shooting pains in the joints, and the inference drawn was that the uric acid which failed to appear in the urine had been retained in the articular structures. The pricking pains were assumed to be of a *quasi* gouty character.

Other forms of neuralgia are not uncommon in gouty subjects; sciatica and facial neuralgia, for example, sometimes alternate with articular gout. Dr. Graves† alluded to the fact that many persons of a gouty habit are subject to sudden pains and twitchings, lasting only a few minutes, or even seconds, and he regarded these symptoms as the results of momentary congestion or of fugitive inflammation. He recognised the existence of gouty neuralgia, especially in the forms of sciatica and tic

* "Uric Acid as a Factor in the Causation of Disease," 1892, p. 4, *et seq.*

† "Clinical Medicine," New Syd. Soc. Ed., vol. i., p. 530.

douloureux. Sir James Paget's statements are much to the same effect. He thinks that gouty neuralgia is distinguishable from other forms by the suddenness and fitfulness of the attacks, and by the readiness with which they are induced by errors of diet, etc. Gouty subjects are especially liable to many varieties of pain and dysæsthesia, such as numbness and sensations of pins and needles in the fingers and toes. As probably of gouty origin, Mr. Hutchinson mentions sharp explosions of lightning-like pains over the parietal bones, and occurring in quick succession, but unaccompanied by tenderness on pressure. Such attacks are sometimes to be traced to indulgence in wine and animal food; they are relieved by purgatives and alkalies. We may assume that the pain is due to hyperæmia and œdema of the neurilemma, but why only certain branches of a nerve should be affected as a result of the constitutional disorder is a question which cannot be solved. Dr. Buzzard in his lectures on "Paralysis from Peripheral Neuritis" points out that when "urate of soda is present in the blood, it may be liable to find its way into the lymph-spaces which are in immediate connection with the bundles of nerve-fibres, and there set up inflam-

mation. The difficulty is to say why this does not always happen, not to explain its occasional occurrence." It is probable that gout is for the most part only a predisposing cause of neuralgia, and that the excitement of the pain is due to the operation of other causes. As a matter of course, all neuralgic pains occurring in subjects with gouty tendencies must not be too hastily attributed to the influence of the diathesis. Dr. Buzzard has drawn attention to the fact that the lightning-like pains of tabes are not unfrequently ascribed to gout or rheumatism. Dr. Graves was of opinion that "gouty inflammation of the nerves and their neurilemma may in process of time extend to the spinal marrow and its investments, and give rise to derangements terminating in *ramollissement* and structural degeneration." He has described several cases of gouty paralysis.* Dr. Angel Money† has lately recorded a case in which many indications of disorder of nerve-centres (*e.g.*, increased tendon reflexes) were referable to a gouty condition. Peri-

* For evidence in support of the view that inflammatory processes in nerves may be propagated to the cord, see Leyden's essay on "Reflex Paralysis," German Clinical Lectures, New Syd. Soc., p. 163. Also Nothnagel's essay on Neuritis in the second series of Lectures.

† *Brit. Med. Journal*, Oct 29, 1887.

pheral neuritis would appear to be the starting-point in cases of this character.

Gout, whether inherited or acquired, may be regarded as a potent cause of certain affections of the eye. Deposits of urates are sometimes visible in the conjunctiva, arranged in minute particles around and along the course of small blood-vessels and their ramifications. Mr. Hutchinson* has arranged the different gouty eye-affections in two groups, according as they are associated with (1) acquired and (2) inherited gout. In the first group, which includes hot eyes, sclerotitis, recurrent iritis and retinitis hæmorrhagica, the attacks are often acute and painful, but generally of a transitory character. In the second group, the attacks are prone to be chronic and persistent, though temporary recovery and recurrence are often observed; the invasion is frequently insidious, but the disease is usually in the end destructive. This category includes insidious disorganising iritis, relapsing cyclitis, certain forms of soft cataract, and perhaps some of primary optic neuritis. Mr. Hutchinson also points out that a

* See Bowman Lecture for 1884, "On the Relation of certain Diseases of the Eye to Gout," *Medical Times and Gazette*, vol. ii., 1884, p. 703.

gouty tendency plays a large part in the production of glaucoma, especially in early life, and that retinitis hæmorrhagica is of very rare occurrence indeed except in those who are themselves gouty. In fifteen cases of retinitis hæmorrhagica, recorded by Mr. Hutchinson, gout was demonstrated in six and probably existed in four or five others. According to Mr. Brudenell Carter,* iritis and keratitis in gouty and rheumatic subjects have a somewhat special tendency to spread to the anterior sclerotic zone. When this feature is observed in a given case, inquiry should always be made as to any gouty tendency. Mr. Carter points out, however, that many cases *supposed* to be of a gouty or rheumatic nature are really instances of glaucoma. It is obvious that an accurate diagnosis, based upon due consideration of all the symptoms and careful examination with the ophthalmoscope, is of supreme importance in such cases.

With regard to the *ear,* tophi are often found on various portions of the pinna. These little masses vary in size and number; some are as large as a split

* See Mr Carter's Article in Dr. Quain's "Dictionary of Medicine," p. 479.

pea or larger. At first they are soft and elastic, and are found to contain a milky or creamy fluid. Later on they become hard and more firmly attached to the cartilage. They are often the seat of pricking pains, which are especially liable to be felt before and during an attack of gout in the toe. According to some authorities, bony growths (exostoses) in the external meatus are frequently of gouty origin. It is doubtful whether the constitutional disorder has any influence in the causation of diseases of the tympanum and labyrinth.

Various disturbances of the sensorial functions often occur in gouty subjects and take the form of vertigo, dizziness, tinnitus aurium, perversions of vision, etc. I frequently attend gouty subjects in whom vertigo is very marked and troublesome. There is, of course, nothing peculiarly characteristic in these symptoms of nervous irritation, for they are common indications of dyspepsia. Sometimes, however, they alternate with sub-acute attacks of articular gout, subsiding when the inflammatory symptoms are prominent, and recurring shortly after their disappearance. Under these circumstances their connection with the gouty diathesis would appear to be unquestionable. Other

manifestations of nervous disorder in gouty subjects, such as cramps in different parts of the body, hysteria, and hypochondriasis, may, in some cases at least, be placed in the same category. The relations between gout and various morbid conditions of the mind were noticed by Sydenham. "His mind is, as it were, affected with the contagion, and so far suffers with the body that it is hard to say which of them is most afflicted."

I have already referred to the fact (see p. 62) that eczema often alternates with symptoms referable to gout, and I have found the association to be of such frequent occurrence that I regard the skin-affection in these cases as a decided manifestation of the gouty diathesis. This view is advocated by many authorities; among others, by my friend Mr. Wyndham Cottle, who informs me that he considers the eczema, in common with the other irregular symptoms, as but a local manifestation of the same constitutional state. Even the late Dr. Hilton Fagge, who was sceptical as to the connection between gout and many other disorders, admitted that a relation probably existed between gout and eczema. "Few cases of eczema are gouty, but those affected with chronic gout are more

subject to eczema than other persons of the same age, and the disease is apt to be particularly irritable, and is more readily curable by alkalies and colchicum."*

This causation of eczema has been minutely discussed by Dr. Piffard,† Professor of Dermatology in the University of New York, and his remarks are so consonant with my own observations that I venture to quote them. In opposition to the Vienna school, he believes in the existence of a certain cutaneous diathesis, which he designates as *rheumic,* this being equivalent to the *dartrous* diathesis of the French, and the *predisposing* cause, he assumes, of eczema, psoriasis, and pityriasis. This diathesis also corresponds with the condition known in England as *lithæmia,* or, in other words, with the uric acid diathesis. The state of the blood in lithæmia has been already fully described, and it is a remarkable fact that uric acid has been detected in the scales and excretions of the skin-affections just mentioned,‡ and that, as described by Sir A. Garrod,

* "Text-Book of the Principles and Practice of Medicine," 3rd Edition, vol. ii., p. 681.

† "A Treatise on the Materia Medica and Therapeutics of the Skin," 1881, p. 126, *et seq.*

‡ In several cases of patients lying bedridden from rheumatic gout, in which one or both legs were covered with an eczematous eruption, the late Dr. Golding Bird observed microscopic crystals of sodium urate scattered like fine hoar-frost on the parts on which the surface-exudation had dried.

the administration of uric acid internally has been followed by an eczematous eruption.* It would therefore seem that these cutaneous affections are really due to accumulation in the blood of certain excrementitious substances, of which uric acid is the one most easily demonstrable. Dr. Piffard thinks that the category of noxious agents includes also lactic acid, oxalic acid, creatin, and creatinin; all of these representing either steps or side-products of the processes which bring about the metamorphosis of food into tissue, and that again into substances ready for excretion. If we regard the normal process as one of oxidation, the production in excess of these excrementitious materials may be assumed to result from sub-oxidation.

So long as these products are removed by the kidneys, there may be no evidences of any injurious action; but when the excess becomes too great, or when the eliminative functions of the kidneys are imperfectly performed, the work is thrown upon other organs, and especially upon the skin, and the result in many cases is an outbreak of eczema, psoriasis,

* Lumleian Lectures, *Medical Times and Gazette*, vol. i., 1883, p. 318.

or pityriasis. These affections obviously present a mutual relationship, and possess many features in common, and they often alternate not only with affections of the joints, but with symptoms referable to the pulmonary and gastric mucous membranes. Eczema is very liable to occur in elderly patients, at a time when the arthritic attacks have become less troublesome. Psoriasis and eczema are often hereditarily transmitted, and in some cases they appear to be the only manifestation of the gouty diathesis which is thus handed down. According to Sir A. Garrod, prurigo must be added to this list. As additional evidence of the connection between these skin-affections and gout, it should be mentioned that they are almost invariably aggravated by causes which increase the production of uric acid; whereas they are improved by constitutional treatment and regimen, but either not influenced at all, or else made worse by ordinary remedies. They are, moreover, very liable to recur so long as the original diathesis remains unchanged.

# CHAPTER VI.

## HEPATIC AND RENAL DISORDERS CONNECTED WITH GOUT.

Hepatic Congestion Frequent in Gouty Subjects—Inflammation of the Biliary Ducts, Jaundice, Etc.—Gouty Cirrhosis of the Liver—Question as to its Causation—Influence of other Substances besides Alcohol—Dr. Budd's Views—Biliary Concretions in Gouty Subjects—Renal Disorders due to Imperfect Assimilation and Hepatic Derangement—Albuminuria as a Symptom—Question as to Physiological Albuminuria—Dr. Senator's Views—Digestion-Albuminuria—Dr. Lockie on Connection of Albuminuria of Adolescence and a Gouty Inheritance—Influence of Albuminuria upon the Kidneys—Question as to Indication of Renal Mischief when "Digestion-Albuminuria" is observed—The "Gouty Kidney" properly so-called, varieties of—Association of Infarctions of Urates with Renal Cirrhosis—Symptoms of Gouty Kidney—Gravel, Calculus, Bladder-Affections, and Diabetes in Gouty Subjects—Gout and Life Assurance.

THE part taken by functional disorders of the liver in the causation of gout has been fully described in preceding chapters. Following out the view so forcibly advocated by the late Dr. Murchison, that *lithæmia* is very often due to functional hepatic derangement, I have sketched, somewhat minutely, the symptoms and

causes of the uric acid diathesis, of which gout is to be regarded as a further development. I have stated my belief that a normal condition of the structure of the kidneys generally obtains during the early stages of gout, and that gouty disease of these organs is a consequence of the irritation to which they are subjected during the elimination of the excess of sodium urate, and other products of defective metamorphosis. It must of course be admitted that renal disease of independent origin sometimes exists previously to any gouty manifestation, and that defective capacity of the kidneys will promote the development and accelerate the course of the disorder. I now propose to consider those hepatic and renal diseases which, so to speak, follow in the train of gout, and may be regarded as more or less direct effects of the dyscrasia.

That the liver is rarely healthy in gout is a point of common observation; but it is at least doubtful whether there are any organic affections of this organ directly chargeable to the gouty diathesis. I have already referred to the symptoms of hepatic congestion often present in gouty subjects; but those are for the most part of a temporary character, are much relieved by proper diet and treatment, and frequently

disappear when the articular inflammation comes on. On the other hand, a paroxysm of gout is sometimes attended by bilious vomiting and purging and other signs of hepatic disorder. Dr. Murchison states that adult persons of a gouty constitution are especially liable to attacks of inflammation of the *biliary ducts*, generally as a result of extension of catarrh of the stomach and duodenum, and preceded by symptoms referable to these parts. In some cases the symptoms of inflammation of the biliary ducts become very severe; the frequent vomiting, the emaciation, and the persistent jaundice have sometimes led to the suspicion of cancer. Under treatment by purgatives, colchicum, and alkalies, the symptoms gradually pass off. This affection is generally noticed in persons descended from gouty ancestors, but presenting no decided manifestations beyond occasional twinges of pain in some of the smaller joints. In other instances the jaundice and other symptoms referable to the liver appear on the subsidence of an acute attack of articular gout. A case of this kind was under my care a few years ago. A lady, aged forty-four, of gouty family history, had had several acute attacks in the right toe-joint; during the intervals she had

suffered much from flatulence and other symptoms of dyspepsia. When called in to see her, I found her suffering from jaundice and considerable pain in the hepatic region. A week previously an acute attack in the joint had suddenly abated, in consequence (as she thought) of a chill, and the hepatic symptoms appeared two days afterwards.

A gouty form of cirrhosis of the liver has been supposed to occur, but the evidence on this point cannot be regarded as quite conclusive. The two conditions not unfrequently co-exist; but their union in such cases is probably due to the operation of the same causes. Trousseau's description of *gouty chronic hepatitis* is exactly applicable to ordinary cirrhosis. He states that it "is characterised by pains in the right hypochondrium; by an increase or diminution in the volume of the liver, rendered appreciable by palpation and percussion; by jaundice, or at least by a sub-icteric tint of the skin. At the autopsy, the substance of the organ is often found exceedingly hard, granular, like cirrhosis, and (according to Lieutaud) charged with calcareous concretions."* The

* "Lectures on Clinical Medicine," New Syd. Soc. Trans., vol. iv., p. 381.

excessive use of alcohol, especially in the form of raw spirits, is universally recognised as the *ordinary* cause of cirrhosis; but Dr. Budd* has suggested "that there may be other substances, among the immense variety of matters taken into the stomach, or among the products of faulty digestion, which, on being absorbed into the portal blood, cause, like alcohol, adhesive inflammation of the liver." This view is certainly deserving of consideration. It is well known that a temporary enlargement of the gland occurs during digestion, and is relieved by an increased secretion and flow of bile. When the congestion frequently exceeds the normal limit (whether the cause be alcohol or articles of food of an irritant nature) it may easily become the starting-point for structural disease. It would seem therefore not improbable that, just as gout may arise from over-supply of food, with little if any excess of alcohol, so hepatic cirrhosis may sometimes own a similar origin. Dr. Sutton† doubts whether the mere abuse of alcohol can give

* "Diseases of the Liver," third edition, 1857, p. 151. Professor Thierfelder doubts whether cirrhosis of the liver ever results from gouty dyscrasia on the ground that the complaint is not common in gouty subjects, and is not attended by deposits of uric acid in the diseased organ. Ziemssen's Cyclopædia, vol. ix., p. 172.

† "Lectures on Pathology," p. 343.

rise to cirrhosis of the liver. He notes a case of extreme cirrhosis in a boy of fifteen, said to be given to drink, though he had not had time to drink much. On the other hand some men will drink enormous quantities of alcohol for many years, and "yet get very little cirrhotic change in the liver." The fact that the disease has been observed in animals (the cow and pig) is sufficient to prove that it is not always due to the use of alcohol.

Biliary concretions are not often noticed in gouty subjects. Dr. Prout, however, thought that a tendency to the formation of gall-stones of cholesterin was frequently associated with a tendency to lithic acid deposits in the urine. This view was supported by Dr. Budd (p. 369). "It is probable that in London the habit of drinking porter, which frequently leads to lithic acid deposits and to the most inveterate forms of gout in persons who inherit no disposition to them, may also frequently lead to the formation of gall-stones." The fact appears to be that gout and biliary calculi often own a similar causation, *e.g.*, errors in diet, sedentary habits, etc. It has been stated that uric acid is sometimes found in gall-stones, and, if such were actually the case, we might infer that a close

relation existed between such concretions and gout. Frerichs, however, states that "uric acid has been found in abundance in a cylindrical concretion described as a gall-stone, but there were some doubts as to the place of origin of this concretion." Stöckhardt and Faber some years ago discovered uric acid in concretions of the same nature. It must not, however, be forgotten how readily concretions in collections are confounded with one another, and wrongly described.* Frerichs thinks that the co-existence of biliary concretions with urinary calculi must be regarded as an entirely accidental circumstance. It is certain that no *special* diathesis is associated with the formation of gall-stones, for they occur in persons of extremely various constitutions, and more frequently in women than in men.

I now proceed to consider those renal disorders which are directly due to the gouty dyscrasia, and I shall first endeavour to trace the influence which imperfect assimilation and hepatic disorder exercise upon their development.

The connection between the urinary secretion and

* "Diseases of the Liver," vol. ii., New Syd. Soc. Trans., p. 497.

disorder of the functions of the liver is shown by the frequent occurrence of albuminuria in certain cases of hepatic derangement. I have noticed this coincidence in not a few patients, men in middle life, of active literary habits, but somewhat free livers. These cases exhibit the ordinary symptoms of gouty dyspepsia, as already described, but no acute attacks of articular inflammation; more or less severe eczema is almost always complained of. The albumen occurs sometimes in very minute quantities, such as one-tenth per cent., while in other cases as much as three per cent. is deposited. No tube-casts are discoverable, and the albumen disappears in the course of a few weeks under the use of suitable diet, purgatives, and alkaline treatment. In no cases of this kind should I think it necessary to prescribe colchicum. I believe that this appearance of albumen in the urine is due to defective metamorphosis of nutritive materials by the liver, and I have little doubt that temporary albuminuria not unfrequently occurs in the absence of any organic disease of the kidneys.

Not many years ago it was commonly believed that the occurrence of albumen in the urine was a decisive proof of the existence of organic disease of the

kidneys. The discovery that albuminuria could be induced by eating a large number of eggs was sufficient to raise doubts as to the truth of this view, and we now have reasons to believe not only that albuminuria may occur in the absence of *renal disease*, but that traces of albumen so often occur in normal urine that this substance may almost be regarded as a regular constituent of that secretion. Cogent evidence in support of this latter view has recently been offered by Professor Senator,* of Berlin. The point is one of great importance, inasmuch as the discovery of albumen in the urine naturally gives rise to the *suspicion* and probably often to the *conviction* that the patient is suffering from some form of Bright's disease. Dr. Senator states that he examined for a lengthened period and at different hours of the day the secretion of four persons, all in excellent health, and at one time or other discovered albumen in the urine of each, certainly only in faint traces, which might well have been overlooked had not delicate tests been employed. "No definite rule governing its appearance could, however, be discovered; for the

* "Albuminuria in Health and Disease," New Syd. Soc. Trans., p. 15, *et seq.*

urine might be examined for several days without once finding albumen, which would then appear for a day and again vanish as before." Dr. Senator cites Leube's * statement to the effect that he found albumen in the urine in 19 instances among 119 healthy soldiers, and also that of Dr. Munn,† who discovered the same condition in 24 out of 200 apparently healthy persons who came before him to be examined for purposes of life-assurance. From these and many other similar experiences, references to which will be found in Dr. Senator's treatise, it would appear warrantable to assume that albumen is often present in normal urine, but only seldom in quantities appreciable by ordinary tests.‡ Sir George Johnson,§ however, while admitting that albuminuria frequently occurs in persons *supposed* to be healthy, maintains that even the occasional presence of the *smallest trace* of albumen is always of pathological import. He alludes to cases in which some obvious cause could

* Virchow's "Archiv.," lxxii., S. 145.

† *New York Medical Record*, March 29, 1879.

‡ See a paper by Dr. Pavy, on "Cyclic Albuminuria" (Albuminuria in the apparently Healthy), *Medical Times*, October 17, 1885.

§ "Latent Albuminuria," *British Medical Journal*, 1879, vol. ii., p. 928. See also "On Intermittent, Recurring, or so-called 'Cyclical' Albuminuria," *Lancet*, 1888, vol. i., p. 7 and p. 999.

be detected, *e.g.*, a previous history of acute nephritis, exposure to cold and wet, as in cold bathing, excessive consumption of food and alcohol, etc. In Dr. Senator's cases, however, there were no histories of this kind.*

It would be foreign to my immediate topic to discuss at greater length this subject of physiological albuminuria: I have alluded to it because it is closely connected with another condition which has been termed the "albuminuria of digestion," and often noticed after the use of large quantities of albuminous food. It is doubtless this form of albuminuria which is often present in cases of gouty dyspepsia; some of the albuminous constituents of the food not being converted into urea, and passing out of the body in an unchanged state. The albumen transudes through the vessels of the glomeruli, and is not a product of the secretion of the glandular epithelium. As a matter of course its presence is most readily detectable when the urine is

* It is perhaps worthy of mention that the mere presence of albuminuria in a gouty subject may give rise to an erroneous diagnosis of kidney-disease, unless due care be taken to ascertain the origin of the albumen. In the case of a friend who suffers from gout and enlarged prostate, the albumen, which can sometimes be detected in the urine, is derived from the increased and altered secretion of the vesical mucous membrane.

scanty and somewhat concentrated, conditions which frequently obtain in these cases of gouty dyspepsia. The subjects of this ailment drink, as a general rule, an insufficient quantity of fluid in the form of water, the place of which in the economy cannot be taken by any other liquid. An insufficient supply of water diminishes the amount of all the important urinary excreta, but facilitates the appearance of "digestion-albuminuria," and especially when at the same time meat and other albuminous foods are taken in excessive quantities. This excretion of albumen, if of frequent occurrence, is very liable to cause irritation of the kidneys. Dr. Senator points out that when egg-albumen as such "finds its way into the blood, it is excreted by the kidneys; but frequently this is not all that happens, for, as Lehmann and Stokvis have observed, *more albumen is excreted than is introduced;* as a matter of course not more egg-albumen, but a form which possesses the properties of the ordinary albuminous substances of the serum (serum-albumin and globulin). It is not improbable that peptone likewise, and perhaps also hemi-albumose (propeptone), may act in a similar way, *i.e.*, excite

albuminuria."* It is worthy of notice, as corroborating the views just expressed, that in cases of albuminuria associated with structural disorder of the kidneys, the urine passed after meals generally contains more albumen than that excreted before breakfast. The effect of exercise is similar to that of the ingestion of food, while rest has an opposite influence. In not a few cases of renal disease, the urine passed in the early morning is quite free from albumen.

In the *British Medical Journal* of June 5, 1886, Dr. Lockie cites four cases of albuminuria, and raises the question as to whether there is any connection between the albuminuria of adolescence and a gouty inheritance. In the cases given there was a decidedly gouty family history, and one main peculiarity of the albuminuria was that it soon disappeared if the patient was kept in bed, and on a milk diet. Headache was a prominent symptom. There was no œdema and no diminution in the quantity of urine. The ages of the patients varied from 12 to 22. They were all members of the cultivated classes, and the albuminuria was especially

* "On the Hygienic Treatment of Albuminuria," New Syd. Soc. Trans., p. 143. See also a paper by Drs. Brunton and Power, "On the Albuminous Substances which occur in Albuminuria," St. Bartholomew's Hospital Reports, 1877.

marked after hard work at school and failure of health. The late Sir A. Clark stated that ten per cent. of the young men competing for places in the Indian Civil Service became the subjects of temporary albuminuria.* Writers on gout and disorders of the liver and kidneys have brought forward many instances in support of the view that, through some failure in preparation either by the stomach or liver, albumen still in a crude state may enter the right side of the heart, and in a condition similar to that introduced in Bernard's experiment, in which crude albumen injected into the jugular vein produced temporary albuminuria. Dr. Sutton† alludes to several causes of functional albuminuria, and quotes Sir W. Gull's opinion with regard to the nature of a case in which the morning urine was loaded with albumen, whereas that passed in the afternoon contained very little, and the urine passed in the evening was quite free. Both physicians came to the conclusion that there was nothing more

* Dr. C. H. Ralfe has recently proposed to apply the term "functional" to that form of albuminuria which is not dependent upon structural disease of the kidneys or proceeding from morbid conditions of the urinary passages. Such functional albuminuria presents at least three varieties, which may be classified as "cyclical," "paroxysmal," and "intermittent." See *Lancet*, 1888, vol. ii., p. 953, and p. 1008. See also a paper by Dr. Moxon, in Guy's Hospital Reports, 1878.

† Lectures on Pathology, page 412.

than functional disturbance. In my own experience I have met with several cases of temporary albuminuria produced by nervous excitement in patients of both sexes, of gouty habit, but without structural change in the kidneys. It may be inferred that the occurrence of albuminuria in young subjects with a gouty family history is more than a mere coincidence.

There would appear to be at least three varieties of functional albuminuria. Those which may be termed the *dyscrasic* forms are caused by changes in the composition of the blood; the *mechanical* form or *cyclical* albuminuria may be referred to alterations in the blood pressure in the kidneys. It is more difficult to account for the *neurotic* forms; but they may perhaps be due to disturbance in the renal circulation. It would seem that all these varieties of presumably functional disorder may ultimately pass into much more serious conditions. Dr. Pavy, however, believes that "functional albuminuria" does not lead to structural disease. It must not be forgotten that the so-called "albuminuria of adolescence" is, in some cases, the result of immoral habits.

A consideration of the facts adduced in the preceding paragraphs will, I think, justify the conclusion that

the defective assimilation which exists in gout may ultimately result in irritation and chronic inflammation of the kidney, and may therefore be regarded as one cause of Bright's disease. Dr. Murchison has strongly advocated this view. He states that when the attack (of acute Bright's disease) follows a chill, and there is no history of scarlatina, "it will almost invariably be found that the patients have previously suffered from derangements of the liver with lithæmia, while many have led intemperate lives. Again we find that functional derangement of the liver resulting in lithæmia, with dyspeptic symptoms such as those which I have described, is a common cause of the contracted, granular, or gouty kidney."

Dr. Dickinson, in the concluding portion of his excellent work on Renal Disorders, has expressed the opinion that albuminuria of dyspepsia is in all probability indicative of renal mischief. "It has been stated that the urine has become temporarily albuminous as the consequence only of dyspepsia; but, knowing as we do how often inconstant or periodic albuminuria together with dyspepsia is the accompaniment of an early stage of the granulating kidney, particularly when this is connected with gout, we can but suspect

that when the urine has become albuminous with symptoms of indigestion, the kidneys may not have been perfectly sound."* With regard to this opinion, it must be admitted that the discovery of albumen in the urine is always sufficient to raise apprehensions of renal lesions; but, when the quantity is small and there are no casts, when there are decided symptoms of gouty dyspepsia, and lastly when the albumen entirely disappears under proper treatment, I cannot but think that we are justified in believing that the kidneys are still sound.† If, on the other hand, the albumen continues, it may not only be indicative of disease, but, as already mentioned, its passage through the kidneys will serve to intensify the condition primarily due to its elimination.

I have met with several cases of Bright's disease in men over fifty, of decidedly gouty tendencies, but never having suffered from acute gout. Sir George Johnson thinks that the granular kidney is often associated with the gouty diathesis, and "is of common occurrence in persons who eat and drink to excess,

* "On Renal and Urinary Affections," Part. III., 1885, pp. 1268-9.

† Dr. Goodhart proposes the term "congestive albuminuria" as applicable to these cases. *British Medical Journal*, 1890, vol. i. p. 1121.

or who, not being intemperate in food and drink, suffer from certain forms of dyspepsia without the complication of gouty paroxysms." After describing the condition of urine (which is at first high-coloured, scanty, and deposits urates, and afterwards pale, more copious, and albuminous), he goes on to say, "in such a case probably renal degeneration is a consequence of the long-continued elimination of products of faulty digestion through the kidneys."* Functional disorder of the stomach and liver, as occurs in gout, may thus result in secondary disturbance of the kidney.

Sufficient evidence has, I think, been adduced in support of the view that the gouty diathesis is closely connected with one form of albuminuria, and this condition may either subside, leaving the kidneys sound, or may by its continuance give rise to organic renal disorder.

I now pass on to consider another form of renal lesion intimately connected with gout, viz., the gouty kidney, properly so called. The morbid anatomy of this con-

* "Lectures on Bright's Disease," 1873, p. 61. See also the paper by the same author on "Latent Albuminuria," *British Medical Journal*, 1879, vol. ii., p. 928.

dition has been already described (see p. 19). Of the two principal varieties, one is characterised mainly by deposits of uric acid and sodium urate scattered throughout the kidney; while in the second, atrophy of the cortical substance and a granular condition of the kidney (renal cirrhosis) are associated with these infarctions.

These deposits and the cirrhotic state of the kidneys are often found in the later stages of gout, but it is certain that these organs are sometimes affected at an early period. The irritation due to the excretion of imperfectly metamorphosed substances may be the starting-point of a chronic inflammation, even before any deposit of sodium urate takes place in the kidney, and it would also seem highly probable that acute gout sometimes attacks the renal fibrous tissue before it appears in the joints. Cases of this kind have been described by Garrod and Charcot. Acute pain in the lumbar regions and temporary albuminuria preceding the articular inflammation, are the principal symptoms; and it may reasonably be inferred that these are due to urate-deposits in the tubules and fibrous tissue.

It must be admitted that some authorities on renal pathology deny that these urate-deposits are neces-

sarily connected with the gouty dyscrasia, mainly on the ground that they sometimes occur in the kidneys of persons who have never exhibited any gouty manifestations. Experience certainly teaches us that cirrhosis of the kidney is far from being peculiar to gout, though it is often found in connection therewith; but with regard to these urate-deposits, especially when found in contracted kidneys, there are strong grounds for believing, with Sir A. Garrod, that they are nearly always indicative of gouty disorder within the affected organs. In gout, according to the same authority, the greater part of the deposit is interstitial; in non-gouty cases, the tubular structure is especially affected, and the crystals of uric acid and sodium urate are larger, but far less abundantly distributed. It is worthy of note that contracted kidneys, exhibiting whitish streaks of sodium urate in the pyramidal portions, are sometimes found in the bodies of persons presenting no external manifestations of gout, but whose joints, when carefully examined, are found to be studded over with chalk-like concretions. Sir A. Garrod refers to cases in which these infarctions were found in the absence of any visible deposits of chalkstones, except one or more specks

on the cartilage of the ear. There are likewise strong grounds for believing that this condition of the kidney is often present in subjects who have suffered from comparatively few attacks of articular gout.

This view of the relation of these infarctions and the cirrhosed state of the kidney to the gouty dyscrasia was strongly advocated by the late Dr. Todd.* In several of his cases the kidneys were found much contracted and wasted at the expense of the cortical substance, the cones in some places reaching almost the very surface of the organ; "in some of the cones there were *opaque streaks of deposit of urate of soda* taking the direction of the tubes, and probably occupying the canals of some of them." Dr. Todd also believed that renal cirrhosis (without infarctions of urates) is peculiarly apt to be developed in the inveterate gouty diathesis, though it may also occur in other states of the system.

From the account given in preceding paragraphs of the state of the kidney often found in connection with the gouty dyscrasia, it would seem advisable to restrict the use of the term "gouty kidney" to those cases in

* "Clinical Lectures," second edition, 1861. Edited by Dr. Beale. See especially Lecture xxviii. on the Gouty Kidney.

which infarctions of urates are present. The expression "gouty cirrhosis" should likewise be applied only to the granular and contracted kidney of *gouty* subjects. Renal cirrhosis often occurs independently of gout, and moreover, in rare instances gout is accompanied by amyloid and other changes in the kidney.

I have lately had the opportunity of observing the development of albuminuria in connection with gout. The patient, a gentleman aged thirty-one, of gouty family, has had periodical attacks of acute gout for the last seven years. The urine has now an average specific gravity of 1012; its reaction is nearly neutral; it deposits on standing a small quantity of muco-pus, and some hyaline casts are visible under the microscope. The quantity of albumen is greater than the muco-pus would yield; it amounts to one-tenth of the test-tube, with the ordinary tests and with picric acid.

I do not think it necessary to describe in any detail the symptoms of gouty kidney. They are for the most part those of renal insufficiency. The urine is generally pale, abundant and of low specific gravity; it contains a diminished amount of urea and salts;

the albumen is usually small in quantity, and at times scarcely detectable. Tube-casts, either granular or hyaline, are almost always present. Nocturnal irritability of the bladder is a common symptom. The patient is generally anæmic and gradually loses flesh and strength, and the symptoms of dyspepsia become aggravated. The pulse is hard and incompressible, and cardiac hypertrophy and dilatation are more or less easily discoverable on examination. Sooner or later there is puffiness of the eyelids, and also slight œdema of the feet and ankles, observable at first only at night; other forms of dropsy, such as ascites and hydrothorax, are less common. As the disease advances, such indications of ill-health as anæmia, debility, shortness of breath, vomiting etc., become more decided, but the progress is often very slow. Attacks of bronchitis, pleurisy, and pericarditis are not unfrequent, and may prove rapidly fatal. Headache and dimness of vision (due to retinitis) are often present; symptoms of apoplexy or epilepsy occasionally supervene, and these signs of uræmia often usher in a fatal termination. In some cases they subside and recur from time to time, the patients feeling comparatively well during the intervals. An

unfavourable termination is, however, always to be looked for, though its advent may generally be retarded by proper treatment.

I shall conclude this account of gouty affections of the kidney by referring to the occurrence of gravel, cystitis, and diabetes in gouty subjects.

Uric acid and urates are frequent deposits in the urine of gouty persons, and as these substances either form or enter into the composition of at least seventy-five per cent. of all calculi, it is reasonable to expect that the subjects of the gouty dyscrasia would be very prone to suffer from calculous disorders. As a matter of fact, gout and calculus sometimes co-exist, and cases are by no means rare in which persons who in early life have suffered from stone in the bladder are attacked by gout at later periods. It must not, however, be inferred that the frequent appearance of uric acid and urates invariably presages the formation of a calculus, for many persons void these substances for lengthened periods, and yet never suffer from stone. I often meet with instances of this kind among persons hereditarily predisposed to gout. The form which the crystals take probably influences the formation of calculi; spiny clumps of uric acid are the most potent in

this respect. Dr. Ord thinks that no calculus is formed of oxalates, urates, and uric acid, or phosphates, without the intervention of some colloid substance, and that the most active matters in forming calculi are the exudation from the tubes and the mucus from the urinary tracts. The question of the origination of calculi* has not as yet been fully solved, and it would be foreign to my present purpose to discuss it any further; I would just remark, however, that calculi of oxalate of lime, or containing layers of this substance, are sometimes found in gouty subjects. I have recently had under my care a lady, aged fifty-six, who has frequent attacks of gout, and occasionally passes small calculi of oxalate of lime. With regard to phosphatic calculi, these of course have no connection with gout, but are for the most part secondary formations due to decomposition of the urine.

Sir W. Roberts † has pointed out that although a special relation exists between uric acid gravel and gout, the two complaints are by no means substantially one and the same. Many gouty people are never

* For an exhaustive account of the latest veiws on this subject, see Dr. Dickinson's work on Renal and Urinary Affections, Part III., p. 860, *et seq.*

† "Croonian Lectures," 1892, p. 56.

troubled with gravel, and many subjects of gravel never suffer from gout. "In both complaints there is an aberration of uric acid; but the error is essentially different in the two cases, both in regard to its site and in regard to its nature. In gout the error occurs on *this* side of the kidneys, in the blood and tissues, and the uric acid is precipitated in a state of combination as a bi-urate; in gravel the error occurs on *that* side of the kidneys, and the uric acid is precipitated in the urine and in the free state." It is true that the one complaint sometimes alternates with the other at different periods in the same individual, or in successive generations of the same family. On the other hand, there is no correspondence between the prevalence of gout and of stone in the several counties of England and Wales.

Irritability of the bladder is a common symptom in gouty subjects, and is generally accompanied by scanty and highly acid urine, depositing uric acid and urates. Sometimes the irritability is attended by increased secretion from the mucous membrane of the urethra and burning pain in this part. I have seen three cases of somewhat severe urethritis, with profuse discharge, in gouty subjects. There was no ground

for suspecting that the symptom was due to any other cause, and the discharge soon ceased after a few doses of alkali had been administered.* In some persons vesical irritability and pain along the urethra occur shortly before an attack of gout. In other cases the vesical irritation accompanies the paroxysm; while in a third class the irritability is relieved or suspended on the outbreak of an acute attack.† Dr. Todd ‡ has recorded a case in which vesical irritation rapidly supervened after an incision had been made into a gouty foot. He has also described cases of spasm and paralysis of the bladder in gouty subjects. Nocturnal irritability of the bladder has already been referred to as a symptom of cirrhosis of the kidney.

It would also appear that inflammation of the mucous membrane of the bladder may be due to gout. Sir James Paget § states that gouty cystitis may present all the ordinary characters of acute inflammation of the bladder from any other cause. Its gouty origin may be suspected if it has begun suddenly and

* Mr. Lawford Knaggs has recorded a case of this kind. *Brit. Med. Journal*, 1891, vol. i., p. 1171.

† See Coulson on "Diseases of the Bladder and Prostate Gland," sixth edition, p. 254.

‡ "Clinical Lectures," second edition, p. 563.

§ "Clinical Lectures and Essays," second edition, p. 377.

after the use of indigestible food, or if it has been preceded by excess of urates in the urine. In some cases cystitis has been known to supervene on the disappearance of an eczematous eruption; Sir James Paget thinks that acute eczema of the mucous membrane of the bladder exists in such cases.

A close connection can sometimes be traced between diabetes and gout. In cases of gouty dyspepsia, the urine frequently contains traces of sugar, and among the sufferers from articular gout, diabetes occasionally supervenes and takes the place of the previous disorder, which is apt to recur when the amount of sugar and the quantity of urine become lessened. The two disorders may, however, co-exist. Sir W. Roberts,* in his account of "Milder Types of Diabetes," mentions a group of cases in which glycosuria occurs in persons advanced in years, of full habit, and with moderate conservation of flesh and strength. The amount of sugar is moderate; uric acid deposits are abundant, and gout is often present. The sugar is sometimes detectible during several years; its quantity varies and the symptom occa-

* "On Urinary and Renal Diseases," fourth edition, p. 308.

sionally intermits. Diabetes occurring in gouty subjects is more amenable to treatment than when this latter dyscrasia is absent. This feature of the complication has been pointed out by Dr. Mortimer Granville.* He emphasises the fact that gouty patients with symptoms of diabetes always require a generous regimen and tonic treatment. Members of gouty families would seem to be peculiarly liable to diabetes; in some of these cases, regular gouty attacks come on, in others there are only slight symptoms. Charcot states that the children of diabetic subjects are predisposed to gout, and cites a remarkable instance of this nature. He mentions also another family in which the father was gouty, while gravel, diabetes, gout, and phthisis occurred among the children. The advocates of the neurotic origin of gout cite the frequent connection between the gouty dyscrasia and glycosuria as evidence in support of their views.

Dr. Hilton Fagge † has pointed out that other facts indicating that lithæmia and diabetes are inversely

* *Lancet*, Oct. 15, 1887.

† "Text Book of the Principles and Practice of Medicine," 3rd Ed., vol. ii., p. 572.

correlated, support the view that both these conditions depend on disorder of the hepatic functions. He alludes to the apparent antagonism between lithæmia and diabetes, and quotes Sir Charles Scudamore's statement to the effect that whereas gout is less common in Scotland and Ireland than in England, diabetes is relatively more frequent in the two former portions of the kingdom. The subsidence of marked symptoms of dyspepsia simultaneously with the appearance of diabetes has been noticed by several observers. The cessation of gouty attacks in persons who have become diabetic may be accounted for by supposing that the accumulation of urates in the blood is checked by the increased flow of urine.

It would be foreign to the purpose with which this book has been written to discuss at greater length the various morbid states more or less closely connected with the gouty taint. Enough, however, has been stated, in this and the preceding chapter, to prove that many kinds of morbid action either owe their origin to this association, or derive their peculiarities from it. A recognition of this fact is of great importance in diagnosis, prognosis, and treatment, and the widespread prevalence of the gouty taint, with its conse-

quences, has a special claim upon the attention of practitioners connected with life-assurance offices. It has been shown by Dr. E. Symes Thompson * that an addition should be made to the age of all those applicants for life-assurance policies in whom there is any trace of gouty symptoms, whether hereditary or acquired. The gouty constitution undoubtedly tends to shorten life, mainly by causing serious lesions of the heart and kidneys.

* "Gout in Relation to Life-Assurance." Read before the Medical Society of London in March, 1879.

# CHAPTER VII.

## THE TREATMENT OF GOUT AND OF VARIOUS DISORDERS CONNECTED WITH IT.

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The Treatment of the Gouty Diathesis—Evidence that the Gouty Diathesis may be Modified or Removed—Objects to be aimed at in the Treatment—Question of Diet for Gouty Subjects—Animal Food—Drawbacks attending excessive use of Vegetable Food—Quantity of Meat to be Allowed—Other Articles of Food—Acids to be Avoided—Farinaceous Food—Alcohol—Milk—Tea, Coffee, and Cocoa—Articles of Diet suitable for Gouty Subjects—Questions as to Gelatine—Quantity of Food—Importance of Diet-Rules—Printed Forms desirable—Production of Sense of Satiety to be avoided—Exercise for Gouty Subjects—Horse Exercise—Walking—Passive Movements—The Zander Method—Fresh Air—Good Influence of Sea Air—Mountain Air—Necessity of Protecting Back and Loins against Cold—Condition of Liver and Stomach—Action of Saline Purgatives—Mineral Purgative Waters, Friedrichshall, Pullna, Condal, etc.—Carlsbad Salts—Other Purgatives and Hepatic Stimulants—Alkalies—Mineral Waters of Bath, Buxton, Wildbad, Teplitz, Vichy, Royat, Baden, Wiesbaden, Harrogate, Aix-la-Chapelle, and Aix-Les-Bains—Woodhall Spa and its use in Gouty Cases—The Sulphur Springs of Dinsdale-on-Tees—Efficacy of Water as a Drink for Gouty Subjects—The Simple Thermal Waters—Alkaline Waters—Muriated Saline Waters—Turkish Baths—Avoidance of Excitement and a proper amount of rest necessary for Gouty Subjects—Treatment of Insomnia—The Treatment of an Acute Attack—Purgatives—Alkalies—Iodine—Salicylate

OF SODIUM—COLCHICUM—BELLADONNA AS A LOCAL APPLICATION—DIET AND REGIMEN—TREATMENT DURING THE INTERVALS—IODIDE OF POTASSIUM—ALKALIES—PIPERAZIN—MINERAL WATERS—GUAIACUM—TONICS AS IRON, QUININE, ARSENIC, ETC.—LOCAL TREATMENT IN CHRONIC GOUT—OPIUM LOCALLY TO RELIEVE PAIN—CHALK STONES AND GOUTY ULCERS—TREATMENT OF GOUTY DISORDERS OF THE LIVER AND KIDNEYS—TREATMENT OF GOUTY DYSPEPSIA AND OF INTESTINAL IRRITATION—TREATMENT OF GOUTY DISORDERS OF THE EYES AND OF CUTANEOUS AFFECTIONS.

I PROPOSE in this, the concluding chapter of my work, to describe the treatment, first, of the gouty diathesis; secondly, of an acute attack of articular gout; and thirdly, of the more important of those disorders which are the direct results of the gouty dyscrasia.

I. *The Treatment of the Gouty Diathesis.*—Experience teaches us that the gouty diathesis may be decidedly modified or even altogether removed, and that these objects can be effected even after several acute attacks have actually occurred. The case of the late Dr. Gregory, of Edinburgh, has been often cited in proof of this assertion. Dr. Gregory was descended from a decidedly gouty family; before he was thirty years of age he had had several severe attacks of acute gout, and occasional gastric spasms due to the same cause. By taking active exercise, avoiding all excesses, and keeping to a strictly moderate diet

(although he did not abstain from animal food) during a period of twenty years, he so completely overcame the disposition to the disease that all symptoms of it disappeared in the latter part of his life. Dr. Todd, in referring to the above case, very justly remarks that "there is no disease in which the patient can do so much for himself, or in which the prescriptions of the physicians are of so little avail without the full and complete co-operation of the patient, as in gout."*

Sufficient evidence has already been adduced in support of the view I have adopted as to the nature and cause of the gouty diathesis, viz., that excessive formation of uric acid is the condition which underlies gouty manifestations of all kinds. It follows that for the rational treatment of this diathesis our object should be to check the production of this *materies morbi* and to promote its elimination from the system. The attainment of these ends is the problem to be solved.

It has been shown in preceding chapters that excessive formation of uric acid and its accumulation in the blood are for the most part due to defective meta-

* "On Gout and Rheumatism," p. 77.

morphosis of the albuminous elements of food, and that the liver is the organ which is mainly at fault. In order, therefore, to combat the symptoms of the uric acid diathesis, the diet and the condition of the liver are the first subjects which require attention.

With regard to the *diet*, an all-important subject in the treatment of any form of gout, I propose only to lay down a few general principles, because in dealing with this disease it is absolutely necessary to make a special study of each patient. Certain maxims, however, apply with greater or less force to all cases. The primary indication is to supply a sufficient amount of those albuminous substances which are readily utilised and metamorphosed in the system.

A certain proportion of albuminous constituents is necessary for the wants of the organism, and those systems of treatment which are based upon almost complete abstinence from *animal* food have never been found serviceable for gouty cases. On the other hand, for patients exhibiting symptoms of asthenia, properly selected kinds of animal food are absolutely essential. In sthenic cases, fish will advantageously replace more or less of the butchers' meat usually consumed. The objection to vegetable diet lies in the

fact that the quantity sufficient to contain the requisite amount of albumen will furnish at the same time an excessive proportion of carbo-hydrates; these being more easily oxidised, and therefore more readily consumed in the system than the albuminous compounds, will, to a considerable extent, spare or prevent the disintegration and oxidation of the latter. It is moreover probable that vegetable albumen less readily undergoes disintegration than albumen of animal origin. In gout the disintegrative changes in the albuminates are arrested, and insufficiently oxidised substances remain in the blood. Under ordinary circumstances, when a diet is taken in which animal food preponderates, a larger amount of oxygen is retained in the system than when amylaceous food is in excess.

The result of the above considerations would appear to be that meat is not to be forbidden to gouty patients; though, as a matter of course, certain restrictions must be laid down as to its use. The following are the chief points to be attended to in connection with the diet:—

1. The quantity of meat to be allowed.

2. The quantity and quality of other articles of food usually taken with meat, and which lessen or prevent the oxidation of albuminous substances.
3. The avoidance of acids and of acid-forming substances, the operation of which is to diminish the alkalinity of the blood and juices of the tissues, and thus to favour the precipitation of the urates and their retention in the system.

The quantity of meat and of albuminates in general to be allowed to subjects of the uric acid diathesis should be in strict proportion to the wants of the system, and this limitation should never be exceeded. Physiology teaches us that about twelve ounces of meat and two pounds of bread are amply sufficient to compensate the daily losses of the system of a healthy man. For gouty subjects a far smaller proportion of carbo-hydrates is advisable, and of these latter substances, sugar and starch, having a greater affinity for oxygen, would appear to be even more prejudicial than fat. The recently discovered product of coal-tar, termed "benzoic sulphinide," or more popularly "saccharin," would seem to be a valuable substitute

for sugar and very suitable for gouty subjects. It possesses no nutritive properties, but its sweetening power is nearly three hundred times that of cane sugar; about one grain is sufficient to sweeten a cup of tea. The experiments of Drs. Stevenson and Wooldridge show that saccharin is quite innocuous even when taken in excessive quantities; that when taken in any reasonable quantity it does not interfere with or impede the digestive processes, and that its continuous use for lengthened periods is not in any way harmful. It does not undergo fermentation, but passes unchanged into the urine, and exercises a sedative and antiseptic action on the mucous membrane of the urinary tract.

For the reasons above given, farinaceous food, such as bread, rice, potatoes, etc., should be used very sparingly; pastry of all kinds should be strictly forbidden. A little fruit may be allowed, provided that no undue amount of acid or of saccharine materials is thereby introduced into the system. Both theory and experience teach us that alcohol in any form should be avoided by the majority of gouty subjects; it undergoes oxidation in the system and checks metamorphosis. Sir Thomas Watson's opinion on this

subject may be quoted with advantage: "I am sure that it is worth any *young* man's while who has had the gout to become a teetotaler. But the case is different with the *old*, and with those whose health has been broken by inveterate disease." Plain pure water, aërated water, or some of the alkaline acidulous waters, form the best fluids for gouty subjects. In cases, however, in which the digestion is feeble, a small quantity of alcohol is sometimes beneficial. Old whisky or brandy is probably the least injurious form of alcoholic stimulant; a tablespoonful diluted with six ounces of water should be the maximum quantity allowed. If wine be preferred, *sound* claret, still moselle, and hock are best suited for gouty patients. One, two, or even three wine-glassfuls may be permitted, according to circumstances; and dilution with some alkaline effervescing water is generally advantageous. Permission to use alcohol in any form should, as a rule, be withheld, unless the patient promises to limit himself to the prescribed quantity, and to take it with or soon after meals. Imperfectly fermented wines, and malt liquors of all kinds, should be strictly forbidden. Effervescing wines, such as champagne and moselle, are generally included in the

list of forbidden liquids. The rule is a good one, but is not universally applicable. In cases of asthenic gout, characterised by multiform symptoms, gradually increasing in severity, and but little influenced by treatment, two or three glasses of sound champagne, allowed daily for as many days, are often very advantageous, and pave the way for special treatment. Dr. Burney Yeo* thinks that the cheap kinds of claret are particularly injurious, and that the *quality* of the wine prescribed is a very important point. Acids and all substances likely to undergo acid fermentation must be interdicted. Acids when absorbed into the blood lessen its alkalinity, inasmuch as they combine with the alkalies they meet with, and set free weaker acids. With the object of preventing scorbutic symptoms, I sometimes allow gouty persons, for whom vegetables would be injurious, to take a little lime-juice, well diluted with water.

There is some difference of opinion with regard to the effect upon gouty subjects of a diet composed largely of milk. When large quantities of milk are taken, the general result is an increased production

* *Lancet*, 1887, vol. i., p. 320.

of uric acid, and this fluid would therefore appear to be unsuitable for such patients. Experience, however, shows that some gouty subjects can take one or two pints of milk in the twenty-four hours with apparent benefit. The prejudicial effects of milk would appear to be due to the lactic acid which results from the fermentation of the milk sugar. It is worthy of note that the administration of lactic acid to gouty subjects causes the attacks to become not only more frequent, but also more severe and obstinate. Moderation should be observed in the use of tea and coffee; the former especially should be avoided whenever flatulent dyspepsia is a prominent symptom. Cocoa is often preferable for these patients. The nibs should be coarsely powdered and boiled for two or three hours; the liquor is then strained, and the fat, which rises to the surface, removed when cold.

From what has just been stated it will be evident that only a few articles of diet can be safely recommended to the subjects of the uric acid diathesis. Mutton, beef, chicken, game, fish, eggs, and green vegetables (including cress, lettuce, etc.), constitute the list of articles from which a selection must be made. A few ounces of stale bread and a small

quantity of butter may be allowed. A diet thus restricted will furnish the necessary amount of albuminates, and the articles are easy of digestion and not likely to set up acid fermentation in the stomach. Substances containing much gelatine should be avoided. Gelatine when introduced into the system appears to undergo a decomposition analogous to that of the albuminous compounds; the ingestion of large quantities is followed by a marked increase in the proportion of urea in the urine, with decided elevation of the specific gravity. In non-gouty subjects the uric acid is not increased in quantity, but it is probable that where metamorphosis is imperfect, other substances, and uric acid among the number, would be formed in place of urea.

It is not sufficient to lay down rules as to the *quality* of the food; the *quantity* is a point of no less importance. I have for some time past been in the habit of supplying my patients with printed "diet-rules." I keep at hand a set of forms on which the hours for meals, and the articles that *may* be taken and those that *must* be avoided are clearly specified. As a matter of course, modifications, by way of addition or subtraction, are sometimes re-

quired, and spaces are therefore left for additions. I attach the greatest importance to these diet-rules; a patient is far more likely to obey instructions contained in a printed form than verbal directions, however emphatically expressed. To the list of articles I have appended a caution with regard to the ill effects of rapid eating. Gouty patients, above all others, should exercise great moderation in the quantity of food taken at each meal. A positive sense of satiety should not be experienced, for, as justly remarked by Dr. Beaumont, "this is beyond the point of healthful indulgence, and is Nature's earliest indication of an abuse and over-burden of her powers to replenish the system." That which ought to be attained "occurs immediately previous to this, and may be known by the pleasurable sensations of perfect satisfaction, ease, and quiescence of body and mind. It is when the stomach says *enough;* and it is distinguished from satiety by the difference of sensations—the latter saying *too much.*" When, as not unfrequently happens, corpulence is associated with gout, the quantity of all articles of diet must be uniformly reduced; that of the albuminous kinds should be considerably dimi-

nished, for no one becomes corpulent without taking albumen, in some form or other, in excess of the requirements.

Exercise and abundance of fresh pure air come next to diet in the treatment of the gouty diathesis. Exercise promotes tissue-change and the oxidation of the constituents of the body, and many a gouty patient of lazy, indolent habits would get rid of his complaint if he were to become a farm labourer or a postman. Walking exercise for several hours a day should, whenever possible, be enjoined. The production of a fair amount of perspiration is generally desirable. Horse exercise, "the Palmerstonian cure for gout," as one German writer terms it, is also very advantageous, and should be tried whenever circumstances will admit. Muscular exercise promotes the consumption to some extent of albuminates, and to a far greater degree of glycogen and sugar. For patients unable either to ride or to take the requisite amount of exercise on foot, some form of gymnastics of an easy kind, and either with or without movable apparatus, may be prescribed with advantage. Mental activity in moderation is also desirable, for it too promotes tissue-change. When patients are incapable of

active exertion by reason of gouty deposits, stiffness, and pain in the joints, some form of passive movement, with friction, shampooing, etc., should be recommended. With precautions adapted to individual cases, some of the methods comprised in Ling's system (the Swedish Movement Treatment) often produce good results. Dr. Zander, of Stockholm, has invented a series of machines by means of which the several muscles of the body can be brought into play, and thus a considerable amount of exercise can be taken without undue fatigue. The amount of exercise, whether natural or artificial, must be *carefully adapted to* the circumstances of the case; anything approaching to undue fatigue must be avoided. Acute attacks may not unfrequently be traced to over-exertion. In connection with this subject it must be kept in mind that a certain interval should always elapse between exercise and meals. Over-fatigue is a fertile source of indigestion, and to sit down to a meal immediately after exercise is not conducive to normal digestion.

Fresh air is especially indicated for gouty subjects; but exposure to damp and cold should be carefully avoided. Sea-air often causes a marked improve-

ment in these cases, and should always be tried. It promotes in a special manner the oxidation and transformation of albuminous tissues. In connection with the subject of change of air as a portion of the treatment applicable to gouty subjects, I would lay stress on the advantages often derived from a short sea-voyage. I have witnessed great benefit, sometimes amounting to a cure, experienced by debilitated subjects after a voyage to the Cape. All the arrangements on board the steamers are most satisfactory. I can speak from personal experience of several voyages to Madeira and back by the "Castle Line" steamers. These vessels are really floating hotels; every attention is paid to the passengers' comfort, and the medical officers in charge are excellently qualified for their posts. Under favourable conditions of weather, the journey is most exhilarating, and the patients' mind and body are alike refreshed and strengthened. The bracing air of mountainous districts is beneficial for many patients; on the other hand, cases of confirmed gout often require a considerable amount of warmth. The clothing of the gouty should be adapted to the season, and in

proportion to the patient's capacity for generating heat. Woollen underclothing of varying thickness should be worn throughout the year, and special care should be taken to ensure well-fitting boots or shoes.

The liability to catarrh evinced by subjects of the uric acid diathesis has been already mentioned (see page 56), and this would seem to be a fitting place for a reference to prophylactic treatment adapted for special cases. Gouty patients, besides suffering from bronchial and naso-pharyngeal catarrhs, often complain of what they term "chills on the liver." In these cases, besides recommending woollen underclothing, male patients should be advised to protect themselves from cold by having their waistcoats and the upper parts of the trousers partly lined with chamois leather. Women suffering from chronic gout often complain of a sensation of cold over the sacral and gluteal regions. In these cases, during exposure to cold, the urine is very apt to be loaded with lithates, whereas it is perfectly clear so long as the patients remain indoors and are protected from chills. By way of prophylactic treatment, some material, such as chamois leather, impervious to wind, should be sewn

on the inside of the corset, and should extend upwards, so as to cover the liver, and downwards to the sacral region.

The effects of deficient protection by clothing are often exemplified among gouty men, whose aches and pains and other uncomfortable sensations are very apt to become troublesome when morning costume is exchanged for the evening dress suit. The front of the body is still tolerably well protected by the double fold of the under-vest, the stiffly-starched shirt front (in itself an excellent non-conductor), and by the cloth of the waistcoat. The back, on the other hand, is covered only by a single fold of the under-vest, the thin, limp linen or cotton of the shirt, and the lining of the waistcoat. The dress-coat contributes but little towards the protection of the back from cold, and the consequences of this defect are the more serious inasmuch as the superficial nerves conducting impressions of temperature are more abundant on the back than on the front of the body. For patients such as those above described, it is well to supplement the defects of evening dress by wearing a thin chamois leather waistcoat under the white shirt. I have often witnessed the good results of thus protecting the back,

and especially in patients suffering from chronic gouty bronchitis. Their symptoms are invariably much ameliorated.

Attention to the functions of the skin is all-important for gouty subjects. I shall have more to say on this topic when describing the various baths that may be used.

In gouty subjects the condition of the stomach and liver, and of the whole intestinal tract, is second in importance only to the question of diet. I have already explained that frequent deposits of uric acid and urates in the urine are generally due to hepatic derangement, for the relief of which it is useless to prescribe merely alkalies and diuretics, remedies which aid the elimination of uric acid, but do not prevent its formation. Various medicines act more or less decidedly upon the liver, and in cases of functional derangement purgatives are very useful. Saline aperients are those which are generally indicated. They act promptly, and not only remove the contents of the bowels, but cause a decided drain from the intestinal vessels and relieve any congestion which exists in the portal system. Opinions differ as to the way in which these medicines produce their purgative effect. Liebig's

theory was that an osmotic current takes place *from* instead of *towards* the circulatory system. Active purgation, however, may be produced in animals by injecting certain saline solutions into the veins. It would appear more probable that these solutions act by preventing the absorption of the secretions which are constantly poured out from the mucous membrane of the intestine, and are taken up by the veins and lymphatics. The salts best adapted for cases of lithæmia are the sulphates of soda and magnesia (the latter I often prescribe in combination with sulphate of quinia), the phosphate of soda and the tartarated soda. These may be given in the ordinary manner, but for many cases the best way of exhibiting them is in the form of some one or other of such mineral waters as Friedrichshall, Püllna, Kissingen, Æsculap, or Hunyadi Yànos. The Condal, a Spanish mineral water from Rubinat, containing as it does an unusually large percentage of sulphate of soda and a very small one of sulphate of magnesia, is admirably suited as a laxative for the gouty. Besides being free from the bitter taste so objectionable in many of these waters, it does not depress the system, and may be continued for a long time with impunity. The dose must be regulated

according to the circumstances of the case; from two to five or six fluid ounces may be required. It is generally advisable to add an equal quantity of hot water, and the medicine should be taken about half-an-hour before breakfast. The quantity should be sufficient to produce one or two evacuations without griping or much discomfort.

When symptoms of gastric catarrh, fermentation, and acidity exist, Carlsbad water is preferable. The principal salts contained in the water of the Sprudel spring at Carlsbad are the sulphate and carbonate of soda and chloride of sodium. Carlsbad salts obtained by evaporating the water can be procured in the solid form, and when dissolved in a suitable quantity of water are very efficacious. About a teaspoonful should be dissolved in half a pint of boiling water, and when the solution has cooled down to about 120°, two or three ounces should be taken every five minutes. This treatment may be continued for several weeks, if necessary; the solution being taken every morning or every other morning, according to circumstances. The quantity of the salt should be increased if the bowels are not sufficiently acted upon; or a small dose

(gr. j.) of the extract of aloes may be taken before dinner.

Other purgatives and certain hepatic alteratives are often useful in the treatment of the uric acid diathesis. Small doses of calomel or blue pill, either alone or combined with colocynth or rhubarb, may be given from time to time. When there is marked congestion of the liver, a full dose of calomel (three or four grains), followed after two hours by an ounce of Mist. Sennæ Co., or a few ounces of one of the saline waters, will be the best plan of treatment. Various other hepatic stimulants, as podophyllin, euonymin, iridin and leptandrin, are available for use in chronic cases. Deposits of oxalate of lime are often found associated with lithates, and when this condition exists the nitro-muriatic acid combined with the tincture of nux vomica and henbane, and taken before meals, will often cause both forms of deposit to disappear from the urine. As a general rule, acids are contra-indicated in gouty cases, but where *chronic* congestion and torpor of the liver exist, a course of nitro-muriatic acid is almost always beneficial. When the urine contains much free uric acid, and exhibits a marked acid reaction, alkalies are generally indicated, and of

these some preparation of potash or lithia is to be preferred. The alkalies facilitate the elimination of uric acid, but, unlike remedies which promote the hepatic functions, they do not prevent its formation.* The citrate of potash is a convenient preparation : a few doses ordinarily suffice to diminish the acidity of the urine.

In addition to those which are more or less purgative in their action, certain mineral waters have a widespread reputation in the treatment of the gouty diathesis. Among the most celebrated are the waters of Bath, Buxton, Harrogate, Woodhall Spa, and Dinsdale-on-Tees, in this country, and of Wildbad, Teplitz, Vichy, Contrexéville, Obersalzbrunn (Kronenquelle), Royat, Baden, Wiesbaden, Aix-la-Chapelle, Aix-les-Bains on the continent. The Bath waters belong to the earthy thermal class, of which they are the only representative in England. Their temperature is high, viz., 117° to 120°. All the arrangements at Bath are excellent, and the place seems likely to regain its pristine reputation. In company with

* Dr. Murchison attributes to alkalies the power of combating the pathological condition which leads to the undue formation of lithic acid. He regards them as promoters of oxidation and disintegration of tissue. It is, however, by no means certain that alkalies (*e.g.*, liquor potassæ) increase the amount of urea in the urine.

Mr. H. W. Freeman (when Mayor) I have personally inspected the Aix-les-Bains massage system of treatment now practised so scientifically there. The massage douche, worked by Aix Douchèurs, the "Berthollet" thermal vapour baths, the inhalation rooms, such as we see at Lippspringe, and the Siegle's spray rooms for atomising, as we find at Marlioz, are all complete and on the best principles. Dr. Douglas Kerr, the author of a "Popular Guide to the use of the Bath Waters," tells me that as compared with its continental rivals, Bath has the advantage of being well adapted for patients during the winter months. Buxton, Wildbad, and Teplitz are simple thermal waters—that is, their natural temperature is high, but they contain only small proportions of mineral matters. The waters of Vichy and Royat are decidedly alkaline, owing to the presence of carbonate of soda; the Contrexéville springs contain lime, soda, and magnesia; those of Baden and Wiesbaden belong to the muriated saline class, chloride of sodium being the principal saline constituent. The Kronenquelle water contains the bicarbonates of lithia, lime, soda, and iron, and free carbonic acid. It is a mild alkaline water. When taken into the stomach the carbonic

acid escapes slowly, and acts beneficially on the mucous membrane. The springs of Aix-la-Chapelle, Aix-les-Bains, and Harrogate yield water containing sulphuretted hydrogen. The water of Aix-la-Chapelle contains also a marked quantity of common salt. All these waters are taken internally in varying quantities, and most of them are also used for baths.

Our English Spa at Woodhall, in Lincolnshire, deserves a more extended trial than has hitherto been accorded to it. It is remarkable for the quantity of iodine which it contains (7½ grains in 10 gallons), being six times as great as that found in any other European spring. It also contains 35 grains of bromine in the same quantity of water. The Woodhall Spa water would therefore seem to be well adapted for those cases of gout and rheumatism in which iodine is indicated, and clinical experience amply confirms this supposition. I have seen several gouty subjects much benefited by the use of the water, which is likewise rich in chlorides. The baths and pump-room have recently been remodelled and furnished with all the best appliances as are found in the largest and best-appointed of the Continental Spas. The

climate and surroundings of Woodhall are also well adapted for gouty patients, and there is ample and satisfactory accommodation for visitors. As an English sulphur spring, Dinsdale-on-Tees is worthy of notice. It was recommended by Sir C. Scudamore as justly rivalling Harrogate, and as being especially suitable for cases requiring the action of aperients, inasmuch as it contains a considerable quantity of sodium sulphate. It also contains much free carbonic acid, and is by no means disagreeable to the taste.

There can be no doubt as to the efficacy of water as a drink for gouty subjects, and as it is generally difficult to persuade people to make use of what is close at hand, it is often expedient to advise a journey and a stay for several weeks at some one or other of these baths. The action of the simple thermal or indifferent waters is neither greater nor less than that of any ordinary pure water heated to the requisite degree. Water taken internally washes out the stomach, augments secretion, promotes the transformation of tissue, removes waste products from the blood, and thus fulfils many of the indications for the treatment of gout. The disease may be kept in check,

and its symptoms mitigated or removed by drinking three or four pints of water daily. Hot water, especially if taken slowly, is rapidly absorbed by the blood-vessels, and is tolerated without difficulty by the stomach.

When used as baths, the simple thermal waters produce effects similar to those of ordinary warm baths, and depending mainly on the temperature of the water, and the time spent in the bath. The skin is softened and cleansed, its circulation is accelerated, and on leaving the bath the action of the cutaneous glands is considerably heightened.

The simple alkaline waters, as those of Vichy, have an antacid and diuretic effect, and their internal use serves to render uric acid more soluble and thus promotes its elimination. It is doubtful whether any saline matters are absorbed when these waters are used for baths. Some observers state that the urine becomes alkaline after a bath of Vichy water. The same result, however, has been noticed after an ordinary warm bath, and is probably due to the fact that an increased amount of acid leaves the system through the cutaneous perspiration.

The use of the muriated saline waters (which contain chloride of sodium) promotes tissue-change, checks acid fermentation, and accelerates the osmotic circulation in the bowels. When used as baths, these waters stimulate the skin and improve its nutrition.

As remedies for gout, little if any permanent benefit can be expected to arise from the mere use of any of the above-mentioned waters, though they are often valuable adjuvants to more rational plans of treatment. A course at such places as Vichy or Buxton will no doubt relieve many of the symptoms, but the improvement will be only of a temporary character. On the other hand, permanent good is likely to result from the internal use of those waters which contain the sulphates and chlorides and a small proportion of the alkalies. These waters are especially indicated in cases in which abundant formation of uric acid is associated with corpulence and signs of plethora. For weakly persons their occasional use will often prove beneficial, but a course of the muriated alkaline waters or of the simple thermal waters is generally more suitable. One advantage connected with a course of treatment by

baths and waters is due to the fact that, while it is going on, patients are generally very careful in obeying all directions with regard to the diet and regimen. In some cases the good habits thus acquired become permanent. Equal results might doubtless be very often obtained under similar treatment and regimen at home; but such a method fails to commend itself to most people by reason of its simplicity and the comparative facility with which it might be carried out.

After a bath of any kind, the skin should be thoroughly dried and well rubbed with a coarse towel. The use of a flesh-brush is also very advantageous. Turkish baths are not suitable for the majority of gouty subjects; they are sometimes useful for young and plethoric persons with rough and coarse skins and deficient perspiration. They are also beneficial in a small number of chronic cases, provided that there is no cardiac complication. They should not be too frequently taken, as they are apt to cause debility.

Avoidance of undue excitement and a proper amount of rest are the last points to which I shall refer in connection with the hygiene of the gouty

dyscrasia. Nervous exhaustion, from any cause, much intensifies the action of the other factors of gout, and all possible care should therefore be taken to prevent its occurrence. Late hours are especially injurious, and the patient's habits as regards sleep should always be inquired into. Too little sleep is as mischievous as too much; it is a fertile source of nervous exhaustion. In all cases of sleeplessness, the physician should direct his attention to the condition of the stomach, liver, and kidneys: he will often find that the discomfort is due to functional disorder of some one or all of these organs. For gouty insomnia, after due attention to the state of the stomach and bowels, paraldehyde, in doses of ɱxv.-lx., and well diluted with syrup, will be found a useful remedy; also sulphonal in 20 grain doses, dissolved in water, may be given with benefit. Where there is much restlessness and excitability, the following combination often proves useful: ℞ Potassii Bromidi ʒj.; Tinct. Cannabis Indicæ ɱxx.; Tinct. Lupuli ʒj.; Tinct. Chloroform. Co. ɱxx.; Aquam ad ℥ij. Fiat Haustus. Half to be taken at bedtime, and the remainder three or four hours afterwards if required. These remedies produce calm sleep, with no unplea-

sant after-effects. Opium is, of course, generally contra-indicated.

I shall add a few more remarks on the medicines suitable for gouty subjects after a brief description of the treatment of an ordinary acute attack.

II. *The Treatment of an Acute Attack of Articular Gout.*—When called to a patient suffering from an acute attack, I invariably examine the urine for albumen. If this latter be absent, and if there be constipation and signs of congestion of the liver, two or three grains of calomel, followed by a draught containing sulphate and carbonate of magnesia, may be prescribed with advantage. Purgatives, and especially calomel, cause the escape of large quantities of bile from the alimentary canal, and thus, according to Dr. P. W. Latham, remove from the system a quantity of glycocine, which, if reabsorbed, would lead to the consequent formation of uric acid. If there be no marked evidence of hepatic congestion, milder remedies will suffice to relieve any constipation that may be present. The saline draught alone, or ten grains of Pil. Colocynth. et Hyoscyami, with perhaps a grain of calomel or a quarter of a grain of Resin. Podophylli, will generally produce a free

action of the bowels. At the same time I prescribe a mixture with Vin. Colchici ♏x., and gr. x.-xx. of some alkaline salt, such as the bicarbonate of potash or soda, the carbonate of magnesia, or the citrate of magnesia or potash. This should be taken four times in the twenty-four hours, and continued according to circumstances. If there be much fever, Liq. Ammon. Acetat. ʒij. may be added to each dose.

For the relief of the pain I find great benefit from the local application of belladonna in the following form :—R Extract. Belladonnæ ʒij., Glycerini ℥ss., Aquæ ʒij. A sufficient quantity of this mixture is placed upon cotton wool and applied to the affected joint, which should be raised upon a pillow and kept in the position most conducive to the patient's comfort. Towards the end of the attack, while the joint is still painful, I have witnessed great relief from placing the limb in a vapour-bath for ten minutes daily. The application of vapour softens and relaxes the cutaneous tissues and causes perspiration. The foot and leg should be afterwards covered up with medicated wool and carefully bandaged. As a matter of course, while active symptoms continue, the patient should be restricted to such articles of diet as arrow-

root, sago, gruel, milk puddings, etc. Seltzer, Apollinaris, or other aërated alkaline water may be freely allowed. When the acute symptoms have subsided, beef-tea, fish, and chicken may be taken in small quantities; the return to ordinary diet should be gradually effected. Rest and care are essential for some days after the subsidence of the paroxysm. The application of leeches, blisters, or cold in any form to the affected joint is always to be deprecated. Cold applications, indeed, are extremely dangerous (see p. 104).

Dr. Mortimer Granville* speaks very highly of iodine as a remedy for internal administration in acute gout. He states that all the symptoms are much relieved under its use, and notably that the quantity of urine is augmented and the urates are freely passed, while the proportional amount of urea usually rises progressively. He gives ten minims of the tincture in combination with chloride of ammonium, chlorate of potassium and glycerine, well diluted. Dr. Granville recommends the same remedy in the treatment of chronic gout whenever the urea is deficient in proportional quantity. My opinion with

* "Gout in its Clinical aspects," p. 43.

regard to iodine (as a remedy in subacute attacks) is in harmony with Dr. Granville's, but I do not prescribe more than ℥iij. v. of the tincture for a dose. I find that it sometimes answers satisfactorily when the iodides are not well tolerated. Given in these small doses it does not appear to produce any unpleasant symptoms. In chronic asthenic cases I sometimes prescribe iodine in combination with cod-liver oil.

Salicylate of sodium is recommended by Sir Dyce Duckworth and Dr. P. W. Latham for gouty symptoms uncomplicated by contracted kidney or albuminuria. The drug, however, is not so markedly efficacious as in cases of acute rheumatism. It should be continued for several days, free action of the bowels being maintained by purgatives, and the diet being adapted to the severity of the symptoms.

With regard to the use of colchicum in gout, this drug has been proved not to increase the elimination of uric acid, and there is no clear evidence of lessened formation of this substance under its use. Results of a different character have, however, been obtained by various experimenters. Thus Dr. J. M. Maclagan,* in

* See *Edin. Monthly Med. Journal*, Dec., 1851, and Jan., 1852, and also a letter in the *Brit. Med. Journal*, Oct. 1, 1887, p. 743.

some attempts to ascertain the physiological action of the drug, found that after its use both urea and uric acid were eliminated from the blood and increased in the urine. He attributes the remedial action of the medicine partly at least to its power of causing these changes, and not to the conversion of uric acid into urea. Sir W. Broadbent has suggested that colchicum checks the process of metabolism antecedent to the formation of this constituent of the urine, and hence nitrogenised waste, other than urea and uric acid, accumulates in the blood and tissues. According to Dr. Lauder Brunton, this drug acts by paralysing the sensory nerves, the motor nerves and muscles being unaffected. Whether this view of its action be correct or not, colchicum certainly exerts a specific influence on gouty pain and inflammation, and the results are clearly not due to its action as a purgative or a sedative. I prefer to use it in small doses (mx.-xv. of the Vinum Colchici) at intervals of four or six hours. It is sometimes useful in chronic gout, but far less frequently than in the acute attacks. For weakly subjects, colchicum is almost always contra-indicated. Dr. Carter,* of Birmingham, believes that the drug

* *Birmingham Med. Review*, Oct., 1887, p. 160.

"should be avoided in all cases attended with debility and cardiac feebleness; and that its use should be confined to early attacks of a sthenic type, and a pulse of distinctly high tension." He considers that other dilators of cutaneous arterioles, such as nitrite of amyl, nitro-glycerine, and nitrite of sodium, are scarcely inferior to colchicum in their power of relieving the pain of acute gout. When aperients are required, I sometimes order small doses of the acetous extract in combination with colocynth and henbane.

In the intervals between the paroxysms much may be done to prevent recurrences, proper diet and regimen being far more efficacious in this respect than any form of medicine. It is well to caution the patient against the use of any so-called specific, however much lauded in advertisements. Colchicum is the active constituent of most of these preparations, and by its action in checking metamorphosis is likely to produce results of a very harmful character. The presence in the blood of a large amount of nitrogenous waste provokes resistance in the arterio-capillary circulation, with high arterial tension and its various forms of risk and mischief.

It is not to be wondered at that gout-ridden persons generally advise their fellow-sufferers to abstain from colchicum. With this preliminary counsel on the subject of self-treatment by specifics, the directions already laid down with regard to food, fresh air, exercise, attention to the functions of the skin, bowels, etc., should be fully explained to the patient, and he should be warned that if these are neglected, neither drugs nor mineral waters will prove of any avail. Provided that due attention be paid to these subjects, various medicines will help to combat the gouty tendencies and to prevent further attacks, or at least to diminish their frequency and severity.

Iodide of potassium is often serviceable for these purposes. I frequently give it combined with quinine and arsenic; it is, of course, especially indicated whenever gout is complicated by lead-poisoning. Its good effects are often well marked in other cases; it may be advantageously combined with bicarbonate of potash or with small doses of Liq. Hydrarg. Perchlorid. in some bitter infusion if the kidneys be sound. In certain cases, and especially for stout persons of full habit, I have found the following combinations very suitable: —

℞ Potass. Bicarb. gr. xiiss.
Sodii Bicarb. gr. x.
Ferri et Ammon. Cit. gr. ij.
Tinct. Nucis Vomicæ ℥v.
Syrupi ʒss.
Aquam ad ℥j. M. Ft. Haust.

To be taken twice daily half-an-hour before food, whilst effervescing, with 17 grains of citric acid dissolved in half-an-ounce of water; or:—

℞ Potass. Bicarb. gr. viii.
Sodii Bicarb. gr. vii.
Ammon. Carb. gr. v.
Sodii Iodid. gr. j. ij.
Syrupi ʒss.
Aquam ad ℥j. M. Ft. Haust.

To be taken as above, whilst effervescing, with the solution of citric acid (gr. xvii. to ℥ss). If required, a grain or two of quinine may be added to the latter, and the nux vomica or the iodide of sodium may be omitted from the alkaline draught.

Various alkaline remedies are generally useful, their main action being to facilitate the excretion of uric acid. Potash and lithia are the alkalies which form the most

soluble salts with uric acid. These should be given for lengthened periods (four to eight weeks) in small doses, and in very dilute solutions. The mineral waters already mentioned may be substituted for these simpler methods of exhibiting the drugs. The ammoniated tincture of guaiacum is a very valuable remedy in many cases of chronic gout. This drug stimulates the action of the skin and kidneys; it is especially adapted for chronic atonic cases. If thought desirable, the iodide of potassium and the bicarbonate of potash may be given in combination with it. Guaiacum should not be given when there are any decided inflammatory symptoms.

About three years ago attention was drawn to a substance called piperazin, as a powerful solvent of uric acid, and I gave it a fair trial in some thirty or forty cases. At first the results appeared to be satisfactory; but increased experience led me to form an unfavourable opinion of the value of the drug. It is true that piperazin unites with uric acid, forming a combination which is much more soluble in water than the urate of lithium. When, however, piperazin is dissolved in urine, the solution has little, if any, action upon uric acid; and experiments have likewise proved that

the drug has no influence upon the formation of the latter substance. Any effect produced by piperazin would seem to be due to its power of rendering the urine alkaline; but its potency in this respect is far less than that of the carbonates of the alkalies, which, moreover, are usually far better tolerated by the system.

Various tonics and stomachics are often required for gouty subjects. Iron is almost always useful whenever there is marked anæmia, and for such cases I generally prescribe either the tincture of the acetate or steel wine with two or three minims of Liquor Arsenicalis in each dose. The state of the bowels should always be attended to when iron is being administered. An atonic condition of the stomach may be improved by quinine and other bitters. Quinine is also valuable in checking fermentation in the stomach and intestines. In asthenic gout arsenic often proves very useful; it is likewise advantageous in many cases of an opposite character after all active symptoms have subsided. It may be conveniently administered in an effervescing mixture as follows: ℞ Liq. Arsenici Hydroch. ʒss.; Acid. Citric. ʒiss.; Tinct. Calumbæ ʒiij.; Spirit. Chloroform. ʒss.; Aquam ad. ℥viij. Fiat Mist. An eighth part to be

taken with ten grains of bicarbonate of sodium while effervescing, twice a day, half-an-hour after meals.

With regard to the local treatment in chronic gout affecting the joints, the application of the tincture of iodine generally affords the most relief to pain and swelling; mercurial plasters may also be tried. When the pain is very severe, it may be much relieved by opium applied locally. Pieces of lint, soaked in a mixture of Tinct. Opii ℥ij., and hot water ℥j., are applied to the affected joints, and covered with gutta-percha tissue. To avoid movement when renewing the applications, a many-tailed bandage will be found very convenient. Gentle friction with various liniments, and carefully applied pressure by means of bandages, are often beneficial. The bandages should not, however, be continuously applied; on the other hand, movement should be encouraged from time to time. Other remedies often prove very useful in subacute articular gout and muscular pains. I frequently prescribe equal parts of the liniments of belladonna and aconite, made into a paste with bicarbonate of sodium and applied to the seats of pain. Another valuable combination is composed of Menthol ʒiiiss.; Chloroform ʒv.; Liniment of Belladonna ʒiv.; and Olive Oil to ℥iii. Œdema of

the limbs may be relieved by friction, warm douches, bandages, and the adoption of an elevated position. It is connected sometimes with phlebitis and sometimes with renal disease, and in either case is apt to be very troublesome. Chalk-stones, when prominent, should be protected from injury. Much good may be done by the continuous application of alkaline solutions, and especially of a solution of carbonate of lithia (grs. v. to ℥j.), as recommended by Sir A. Garrod. A piece of lint is soaked in this fluid and applied to the joint, and then covered with oiled silk or gutta-percha tissue. The addition of an equal quantity of iodide of potassium increases the efficacy of the solution, especially when there is much inflammatory thickening round the joint. Under similar circumstances, warm douches, passive movements, shampooing, etc., are likely to be serviceable. The removal of chalk-stones, through incisions into the skin, is fraught with danger; erysipelas is very likely to supervene. Gouty abscesses may be very carefully punctured; if ulcers form, they should be protected from injury, and treated in the ordinary way.

III. *The treatment of Gouty Disorders of the Liver and Kidneys and of Gouty Cutaneous Affections.*—

Gouty inflammation of the biliary ducts should be treated by poultices to the abdomen and warm baths, with a little blue pill and the sulphate of soda or magnesia as a purgative. Alkalies, such as the citrate or bicarbonate of potash, are afterwards indicated. The affection of the biliary ducts is generally preceded by catarrh of the stomach and duodenum. The diet must be carefully regulated.

Purgatives are usually indicated for patients liable to biliary calculi. The sulphate of soda may be taken in doses of ʒj.-ʒij. in some bitter infusion every morning. If the bowels act regularly, alkalies, such as bicarbonate of soda, in doses of gr. xx.-xxx., with Succus Taraxaci and Infus. Gentian. may be given twice daily. Durande's remedy for gall-stones consists of ether ʒiij., oil of turpentine ʒij.; of this ♏ xv.-xxx. are to be taken twice daily for a lengthened period.

Gouty dyspepsia is best treated by mild purgatives, such as the Carlsbad salts, and by effervescing alkaline mixtures. The subjoined formulæ are the most useful of those which my experience leads me to recommend: ℞ Sodii Bicarb. ʒiij.; Magnes. Sulphat. ʒiij.; Syrup. Zingib. ℥j.; Aquam ad ℥viij. Fiat Mist. A

sixth part to be taken three times a day, while effervescing, with a sixth part of the acid solution: ℞ Acid. Citric. ʒij.; Aquæ ℥vj. When the dyspepsia is accompanied by decided indications of gout, and the urine is scanty, I often prescribe: ℞ Sodii Bicarb. gr. 320; Magnes. Sulphat. ℥iss.; Vin. Colchici ʒij.; Tinct. Gentian. Comp., Sp. Ammon., Aromat., Tinct. Zingib., aa ℥j.; Aquam ad ℥xvj. Fiat Mist. Take two tablespoonfuls in a tumbler of cold filtered water, one hour before luncheon and dinner daily. Quassia or Calumba may be substituted for the Gentian. For gouty dyspepsia, accompanied by severe aching pains in the muscles and joints, the following formula will prove useful: ℞ Sodii Salicylat. ʒj.; Potass. Iodid. gr. 40; Liq. Arsenicalis ♏ xij.; Sp. Ammon. Aromat., Tinct. Zingiberis, aa ʒiv.; Tinct. Lupuli ℥j.; Aquam ad ℥viij. Fiat Mist. Take an eighth part in half-a-tumbler of water an hour before the last meal daily. When the dyspepsia is associated with constipation, I often order with benefit a mixture as follows: ℞ Mag. Sulph., Sodæ Sulph. aa ℥j.; Sp. Am. Ar. ʒiss.; Pot. Bicarb. ʒj.; Tinct. Nucis Vom. ʒj.; Sp. Chloroformi ʒj.; Tinct. Card. Co. ʒiss.; Aquam ad ℥x. Fiat Mist.

Take a sixth part one hour before breakfast when required. With regard to diet, meat should be allowed only once daily, and alcoholic liquors, sugar, and starchy articles of food should be forbidden. There is no objection to stewed fruits, prepared as suggested by Dr. Milner Fothergill, viz., to one pound of fruit add as much potass. bicarb. in powder as will lie on a shilling. The alkali will neutralise the vegetable acids, and help to bring out the natural sweetness of the fruit. The hygienic treatment of the uric acid diathesis must of course be followed out.

For the relief of symptoms of intestinal irritation in chronic cases of gout, salol often proves very efficacious. It may be given in the form of tabloids, each containing five grains. Within an hour after taking even one tabloid, the urine exhibits the salicylic reaction with persalts of iron. The drug is especially indicated in intestinal gout, accompanied by much flatus, and whenever a tendency to diarrhœa is prominent in gouty subjects. For acute gouty intestinal attacks, characterised by violent colicky pains in the cæcum, nausea, etc., the salicylate of sodium should be given in doses of gr. xv. every four hours until a drachm or a drachm-and-a-half has been

taken, and then every six hours. Dr. Haig states that alkaline salts of sodium and potassium should not be combined with the salicylate, and that the latter should be continued for at least five or six days after all pain is gone and the temperature is normal.

Cirrhosis of the liver occurring in gouty subjects requires the ordinary treatment. With regard to diet, it is not enough to prohibit the use of alcohol, inasmuch as in all probability the disease is sometimes due to the action upon the liver of other irritants, such as ill-digested food, etc.

In the treatment of gouty disorder of the kidneys, the urine should be carefully examined for albumen, tube-casts, and sugar. If the secretion be found normal in these respects, any symptoms referable to the kidneys, such as pains in the loins, frequent micturition, and a highly concentrated and acid state of the urine, will be best treated by saline purgatives, followed by citrate of potash in an effervescing solution. Warm baths, warm fomentations, or mustard plasters to the loins will relieve the pain, and any renal congestion that may be present.

In cases in which there is a tendency for the uric acid to be precipitated within the kidneys, it is advisable

to give a dose of alkali sufficiently large to prevent, as far as possible, such a deposit. According to Sir W. Roberts, the best remedy for this purpose is the citrate of potassium, in half-drachm doses; as a general rule, it is well tolerated by the stomach. One such dose, given the last thing at night, will often greatly lessen the frequency of micturition.

If the urine contain albumen, but no casts, the significance of the albumen is the question to be decided. As mentioned in a previous chapter (see p. 146) albuminuria of this kind not unfrequently co-exists with symptoms of gouty dyspepsia, and is due, I believe, to malassimilation of food. In addition to the remedies already indicated for this symptom, I can strongly recommend *dry cupping* over the loins, repeated, if necessary, from time to time. The albumen rapidly diminishes under this treatment; but its presence must not on that account be regarded as an unimportant symptom, for if neglected it may set up serious disease of the kidneys.

If the albuminuria recur from time to time in a gouty subject, and if casts of the tubes appear, there can be no doubt as to the existence of a renal lesion. The albumen may be in very small quantities, and at

times even absent; the casts are either hyaline or granular. Other symptoms, already described, gradually supervene, and the disease generally runs a protracted course.

The treatment is that of the cirrhotic form of Bright's disease; special attention must of course be paid to the dyscrasia to which the renal affection is due. The measures recommended for dealing with the gouty dyscrasia and for chronic gout must be rigorously enforced. Suitable diet, exercise short of fatigue, a proper amount of rest, attention to the state of the bowels, and to that of the skin, will tend to delay the advent of untoward symptoms. Counter-irritation to the kidneys and hot air baths or vapour baths are valuable remedies in this class of cases. Alkalies are generally indicated, but should be used with caution in cases in which the eliminating powers of the kidneys are defective. Iodide of potassium is sometimes useful, and should always be given when lead-poisoning is suspected. Opium in any form is of course quite out of place. Patients with this condition of the kidneys are peculiarly susceptible to the action of this drug. When the disease is well marked, a nourishing diet and iron are generally

indicated. Warmth and rest of body and mind are absolutely essential.

Cardiac disorders occurring in gouty subjects will of course require various modifications of the ordinary treatment. As a cardiac tonic in gouty cases, the following prescription will be found useful:—℞ Pulv. Digitalis gr. iv.; Extract. Nucis Vom. gr. iv.; Extract. Gentian. gr. xij.; Fiat pil. xij. One pill to be taken twice or thrice daily for five or six weeks. The addition of arsenious acid, gr. $\frac{1}{50}$, to each pill often proves advantageous.

My friend Mr. Critchett has kindly given me his experience of the treatment of gouty conjunctivitis. He relies chiefly on constitutional and dietetic treatment, and states that astringent remedies, such as sulphate and acetate of zinc, invariably do harm, and that in many instances the symptoms are aggravated both by atropine and opium. The local applications which are most likely to afford relief are weak solutions either of lead or of boric acid.

The treatment of gouty cutaneous affections can be summed up in a few words. Eczema and psoriasis always require constitutional remedies; the local treatment is of subordinate importance. The diet

and regimen should be carefully attended to; over-indulgence in animal food should be strictly prohibited, and any co-existing dyspepsia must be dealt with as already described. When the skin-affection is associated with plethora, saline purgatives and alkalies are indicated; when there is debility, the digestion should be aided by vegetable bitters and other tonics. Remedies which stimulate the hepatic functions are generally serviceable. The action of the skin should be promoted by vapour baths or hot air baths. Arsenic may be tried should the skin-affection prove very obstinate, and in cases in which this remedy is found inefficacious, I have seen great benefit result from the use of antimony in doses of 10 to 15 drops of the wine three times a day. The following combination has also proved very serviceable in many of my patients suffering from gouty eczema and erythema: ℞ Potassii Acetat. ʒss.; Tinct. Nucis Vomicæ ʒss.; Infus. Quassiæ ℥j.; Tinct. Chloroform. Co. ♏ xx.; Aquam ad ℥viij. An eighth part to be taken night and morning, fasting. As local remedies, in the early stage of eczema, a lotion containing bromide of potassium (grs. xv. to ℥j.) will often relieve the itching; in later stages, the white pre-

cipitate ointment, zinc ointment, and various preparations of tar are likely to be serviceable. In cases of gouty itching and soreness about the anus, I have found zinc ointment with a drachm of spirits of wine to the ounce most useful in relieving the irritation. Another very useful application, and one I often employ, is an ointment of boric acid (Martindale) composed as follows: paraffin 5 parts, vaseline 10 parts, and boric acid (in fine powder) 3 parts. The following combination often affords marked relief in severe cases of anal pruritus: ℞ Hydrarg. Subchlorid. ʒij.; Bismuth. Subnitrat. ʒij.; Cocainæ Hydroch. gr. vij.; Vaselin. ad ℥j. Fiat unguent. To be smeared over the part and inserted within the sphincter night and morning. It must be borne in mind that eczema in gouty subjects is very apt to recur, especially in the spring of the year.

# INDEX.

C.

D.



E.



F.



G.

## H.

M.



N.

O.



P.



R.



S.

T.



U.

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